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BONE CONDUCTION CHANGES IN ACUTE OTITIS MEDIA

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DESCRIPTION OF THE CLINICAL MATERIAL

An analysis of the results of bone conduction tests is presented here. The tests were performed on 30 patients. Of this number, 25 patients were affected with purulent perforative otitis media and 5 patients with acute congestive otitis media. Of those with purulent disease, 7 had their first inflammatory aural attack and 23 had an acute exacerbation of chronic purulent otitis media. The 5 patients suffering with acute congestive otitis media all had their first inflammatory aural attack during the period under study.

TABLE 1.—*Patients Grouped According to the Acute Changes in Both Ears*

Group	Changes	Patients
1	Bilateral purulent perforative otitis media, acute or exacerbated	5
2	One ear, purulent perforative otitis media (acute or exacerbated); the other ear, acute congestive otitis media	3
3	One ear, purulent perforative otitis media (acute or exacerbated); the other ear, no acute changes	17
4	One ear, acute congestive nonperforative otitis media; the other ear, no acute changes	5
		30

The group represents all consecutive cases of acute purulent and chronic purulent exacerbated otitis media in which satisfactory observations could be obtained. However, it does not represent all such consecutive cases of acute congestive otitis media. Only such cases of the latter type were included in which during the period of the sickness such phenomena of bone conduction were observed as were found to be typical for the purulent, perforative type of middle ear disease.

Both ears of each patient were examined and audiometrically tested, even if the acute lesion was on one side only. According to presence or absence of pathologic changes in both ears, the 30 patients were divided into four groups, listed in table 1.

The grouping of patients described in table 1 results in the grouping of ears indicated in table 2. The age distribution of the patients is given in table 3.

METHOD OF TESTING

On the 30 patients, 87 bone conduction tests were performed. Except in 4 instances, in which the diseased ear only was retested, all tests were performed bilaterally. This gives a total of 193 bone conduction tests performed on 60 ears. The numbers of observations on the individual patients are indicated in table 4.

TABLE 2.—*Ears Grouped According to Acute Changes*

Group	Changes	Ears
1	Purulent perforative otitis media, acute or exacerbated	30
2	Acute congestive nonperforative otitis media	8
3	No visible acute changes in the middle ear	22
		60

TABLE 3.—*Patients Grouped According to Ages*

Age Group	Patients
10-19 years	4
20-29 years	9
30-39 years	8
40-49 years	4
50-59 years	4
60-69 years	1
	30

TABLE 4.—*Number of Bone Conduction Tests Made on Each Patient*

Tests per Patient	Patients	Tests per Group
1	8	8
2	4	8
3	9	27
4	5	20
5	2	10
7	2	14
	30	87

Whenever possible, a complete examination of both ears was obtained in at least two periods, i. e., during the acute stage and again after complete disappearance of inflammation. By this method the hearing in the diseased ear could be compared with the hearing of the same ear when healed. This eliminated reference to audiometric "normals," which are averaged observations and do not coincide with the thresholds of individual healthy ears. However, a certain number of patients were included in this study in whose cases the test was performed only once. These patients all had unilateral disease, and the inflamed ear was compared with the other, healthy ear.

The tests were performed with the 2-B Western Electric audiometer. Its bone conduction receiver is type D-80904. The testing room had no outside walls, was located in a residential section and contained usually about 20 decibels of extraneous noise.

The bone conduction tests were regularly supplemented by air-conduction tests and tragus [1] tests. The Weber test was also performed on every patient of this group, but the results will not be discussed here.

In this group 25 inflamed ears and 10 contralateral healthy ears were also tested by the Bárány modification of the Gellé test.[2] If in the Gellé test the hearing was decreased by both suction and pressure in the external meatus, the test was recorded as positive. If the hearing was increased by the same two manipulations, the test was recorded as inverted and marked by the letter "i." If the hearing was not changed by the variation of pressure in the meatus, the response was designated as negative. It was assumed in this study that a positive or an inverted Gellé test is an indication of a mobile stapes.

TYPICAL MOVEMENT OF THE BONE CONDUCTION CURVE

When the bone conduction curve obtained from an acutely inflamed ear was compared with the curve obtained from the same ear after the inflammation had subsided or healed, certain "typical" phenomena were observed in a great majority of the cases. Chart 1 shows such typical audiograms of 4 patients, belonging to the four groups, respectively, of table 1.

Audiogram 1 (case 1) indicates the hearing of the right and audiogram 2 that of the left ear of Mrs. J. R., who was affected with bilateral acute exacerbation of chronic purulent perforative otitis media. The air conduction and bone conduction curves dated Jan. 21, 1937 were recorded during the acute inflammatory stage, which had started four days earlier in both ears. The curves dated Dec. 20, 1939 were recorded twenty-two months after the discharge and all other inflammatory symptoms had disappeared.

The patient in case 2 had had acute congestive otitis media in the right ear (audiogram 3) and acute purulent perforative otitis media in the left ear (audiogram 4) for about ten days when tested Feb. 4, 1939. With this record is compared one of February 24, when the right ear drum had a normal appearance and the left ear drum was still pink and retracted, but the discharge had ceased and the fistula had been healed for thirteen days.

The patient in case 3 when tested Jan. 8, 1937 had had acute purulent perforative otitis media on the left side for two weeks (audiogram 6).

1. Hůlka, J. H.: Testing of the Transmission Apparatus with the Bone Conduction Receiver, Ann. Otol., Rhin. & Laryng. **48**:1020 (Dec.) 1939.

2. Sonnenschein, R.: The Functional Testing of Hearing, in Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1930, p. 486.

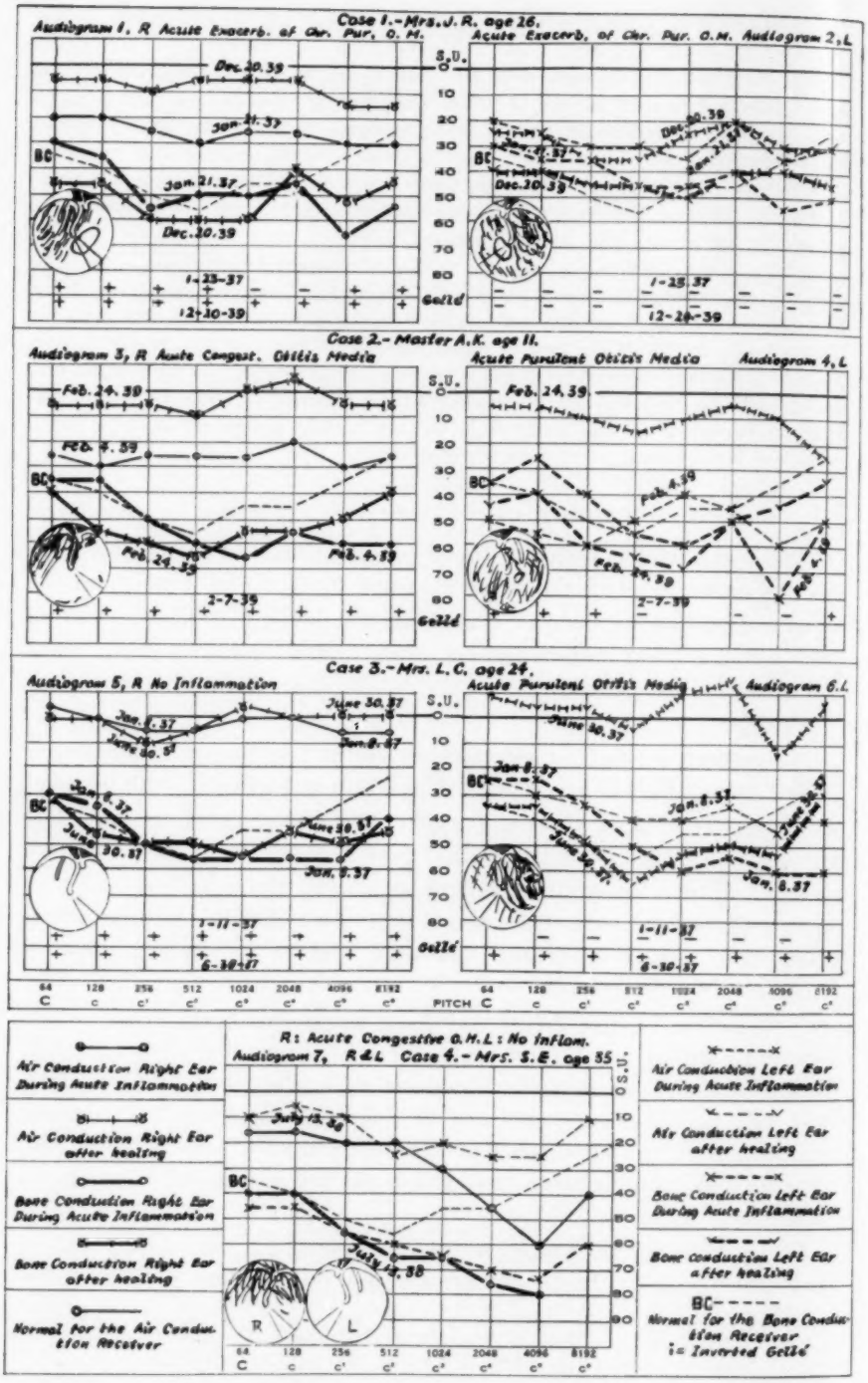
Case 1.- Mrs. J. R. age 26.
Audiogram 1, R Acute Exacerb. of Chr. Pur. O. M.
Acute Exacerb. of Chr. Pur. O.M. Audiogram 2, L
S.U.
BC
Dec. 20. 39
Jan. 21. 37
1-25-37
12-20-39
Gellé
Case 2.- Master A.K. age 11.
Audiogram 3, R Acute Congest. Otitis Media
Acute Purulent Otitis Media
Audiogram 4, L
Feb. 24. 39
Feb. 4. 39
2-7-39
Case 3.- Mrs. L.C. age 24.
Audiogram 5, R No Inflammation
Acute Purulent Otitis Media
Audiogram 6, L
June 30. 37
Jan. 8. 37
1-11-37
6-30-37
64 128 256 512 1024 2048 4096 8192
C c c' c² c³ c⁴ c⁵ c⁶
PITCH
Air Conduction Right Ear During Acute Inflammation
Air Conduction Right Ear after healing
Bone Conduction Right Ear During Acute Inflammation
Bone Conduction Right Ear after healing
Normal for the Air Conduction Receiver
R: Acute Congestive O.M. L: No Inflam.
Audiogram 7, R & L Case 4.- Mrs. S. E. age 35
July 13. 38
July 14. 38
R
L
Air Conduction Left Ear During Acute Inflammation
Air Conduction Left Ear after healing
Bone Conduction Left Ear During Acute Inflammation
Bone Conduction Left Ear after healing
BC
Normal for the Bone Conduction Receiver
= Inverted Gellé

Chart 1.—Typical movements of the bone conduction curves in cases of acute inflammatory otitis media.

No inflammation was visible in the right ear (audiogram 5). The disease was protracted, owing to a granuloma which developed from the promontory in the neighborhood of the round window early in February. The curve of June 30, 1937 was taken two months after the discharge had disappeared and the granuloma absorbed. Both ear drums had a perfectly normal appearance at that time.

Audiogram 7 belongs to the patient in case 4, who had on the right side acute congestive otitis media, which had developed as a complication of homolateral acute purulent antritis. The record for the inflamed right ear is compared in the same audiogram with that for the left ear, in which no inflammatory symptoms were visible. The sickness started about one week before the test was taken.

TABLE 5.—*Bone Conduction Movements During the Acute Inflammatory Stage*

Type of Movement	Hearing of: Low Tones	Hearing of: High Tones	Ears	Pathologic Condition
	1. Atypical Ears			
I	Increased	Increased	2	Suppurative
II	Increased	Not changed	1	Suppurative
	Increased	Not changed	1	congestive
III	Decreased	Not changed	1	Suppurative
IV	Decreased	Decreased	3	Suppurative
	Total number of atypical ears.....		8	
	2. Typical Ears			
	Increased	Decreased	22	Suppurative
	Increased	Decreased	7	Congestive
	Total number of typical (see-saw) ears		29	
	Not checked		1	Suppurative
	Total number of ears.....		38	

The striking feature of all the audiograms of chart 1 was the reciprocating, or see-saw, motion of the bone conduction curves in the diseased ears. During the acute inflammatory stage the hearing of the low tones through the bone increased, while that of the high tones decreased. After healing the low tone end of the curve went down, while the high tone end went up. This phenomenon was observed in regard to 22 of the 30 suppurative ears and 7 of the 8 congestive ears. Was this motion caused by one factor, which acted on one end of the curve only, causing an opposite effect on the other end, or were there two factors, each acting independently on its tone range? Was such a factor (or factors) tympanic or cochlear or both?

ATYPICAL MOVEMENTS

Table 5 lists the ears which did not show the "typical" see-saw movement of the bone conduction curve and indicates which changes

were observed in bone conduction during the acute stage as compared with bone conduction after healing or improvement.

Many atypical movements were also found in curves for the typical ears during the course of the disease, i. e., before the final typical movement occurred. Some of them are represented in chart 2. Audiogram 6b shows 4 bone conduction tests recorded for the left ear of the patient in case 3. Curves 1 and 4 are identical with the curves dated Jan. 8, 1937 on audiogram 6 of chart 1. The relative position of these two curves shows the typical see-saw movement. Curve 1 was taken during the acute stage and curve 4 after healing. On the other hand, curves 2 and 3 represent type I of the atypical movements (see table 5). Curve 2 was taken while the inflammation was still very active; the discharge was copious, and a granuloma, derived from the mucosa of the middle ear, was bulging through a perforation in the posterior segment of the drum head. Curve 3 was registered five weeks after the granuloma and the discharge had disappeared and when only the flaccid membrane remained congested. By comparing on the same audiogram curve 1 with curve 3, or curve 2 with curve 4, one may notice type II of the atypical movement. In each pair chosen for comparison one curve belongs to the suppurative and the other to the nonsuppurative stage.

Audiogram 10b represents bone conduction of the left ear of the patient in case 5 and has features similar to the just discussed audiogram 6b. It shows two bone conduction records (curves 1 and 2) made in the suppurative stage and two (curves 3 and 4) made in the nonsuppurative, healing stage. When the latter curves were recorded, the drum membrane was still congested, and it was more so when curve 3 was taken. Curves 1 and 2 were taken at the same visit, curve 1 before inflation of the eustachian tube and curve 2 after inflation with benzedrine vapors. Between the curves of this audiogram there is observed one atypical type of movement which is not listed on table 5: Movement of curve 2 against curve 4 can be designated as type V of the atypical movements, the hearing of low tones during the suppurative stage being unchanged and that of the high tones decreased.

In audiogram 12b, which has a similar selection of curves (1 and 2 in the suppurative stage, 3 and 4 in the healing stage) one again sees combination 1-4 giving the typical see-saw movement, while combination 1-3 gives type I or type II movement, and so on.

The histories of patients 5 and 6 follow.

CASE 5 (audiograms 9, 10a and 10b).—Mrs. K. M., aged 53, when she was tested on Jan. 23, 1939 gave a history of a blood-stained discharge from the left ear, which started about sixteen days before, after an attack of influenza. Since the appearance of the discharge she had experienced vertigo and had heard noises in the diseased ear. The right drum head was retracted and contained a small centrally located scar. During the whole period of observation no congestion

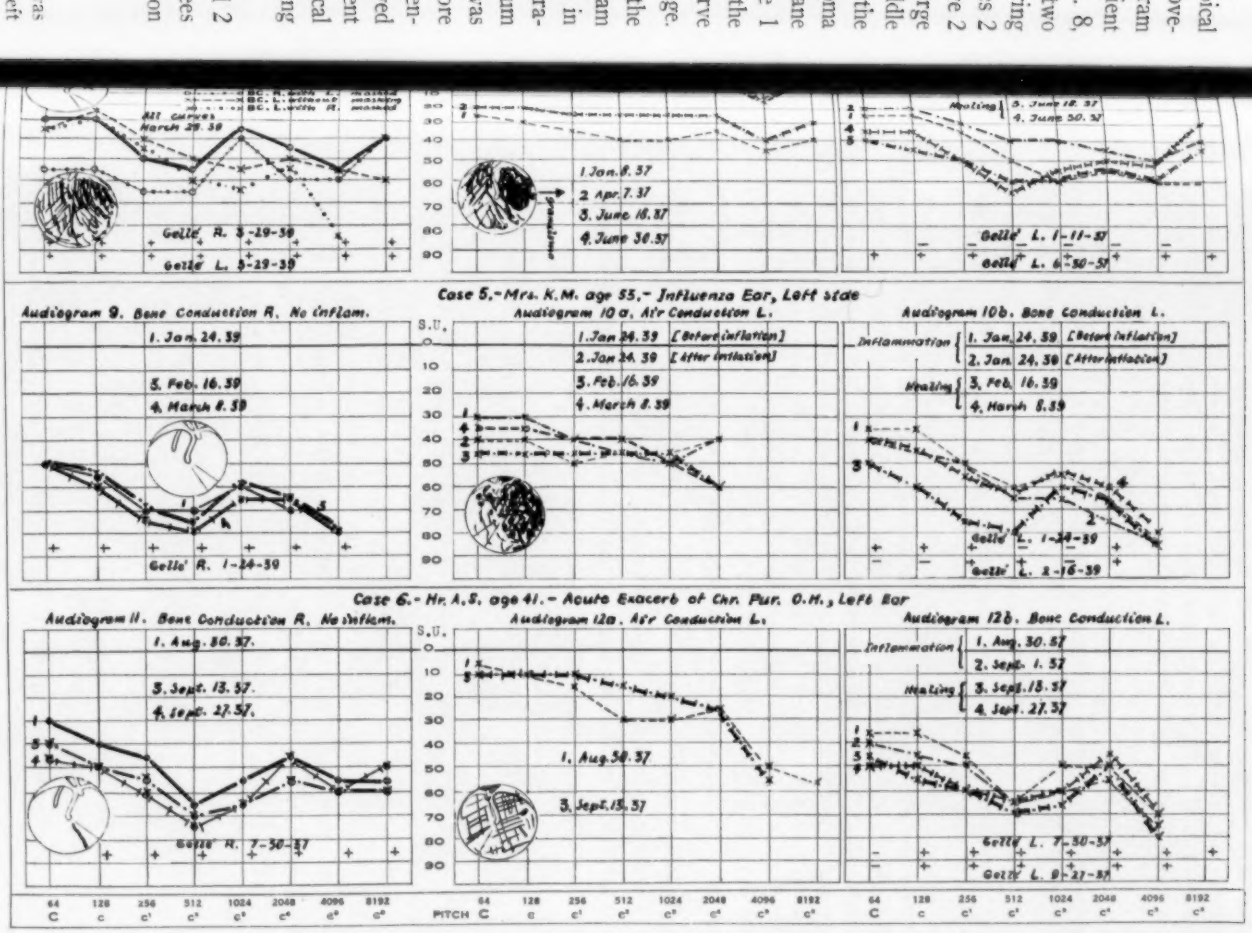
Case 5.- Mrs. K.M. age 55.- Influenza Ear, Left side
Audiogram 9. Bone Conduction R. No inflam.
Audiogram 10a. Air Conduction L.
Audiogram 10b. Bone Conduction L.
Case 6.- Mr. A.S. age 41.- Acute Exacerb of Chr. Pur. O.M., Left Ear
Audiogram 11. Bone Conduction R. No inflam.
Audiogram 12a. Air Conduction L.
Audiogram 12b. Bone Conduction L.
S.U.
PITCH
C

Chart 2.—Atypical movements of bone conduction curves 2 and 3 which occurred in inflamed (left) ears, audiograms 6b, 10b and 12b, before the final typical movement 4 was observed. The corresponding air conduction curves of audiograms 6a, 10a and 12a show that the bone conduction changes cannot always be explained by the noise masking theory. Audiograms of the contralateral noninflamed ears, 9 and 11, show that the patients retaining their pattern were good observers. Audiogram 5a demonstrates effects of transcranial sound transmission in bone conduction tests performed without the use of a masker.

was noticed in this ear. The left membrane was tense and was perforated in the upper and posterior segment. It contained subepithelial hemorrhages, which extended into the epidermis of the meatal wall. The discharge was purulent and sanguinolent. The right eustachian tube was patent; the left, stenotic. The nasopharyngeal mucosa was thickened. The condition was diagnosed as "influenza ear."

On February 16 the perforation was healed, and there was present only a small amount of moist congestion on the flaccid portion. The tinnitus was reduced; the vertigo had subsided. The nasopharynx had a normal appearance.

CASE 6 (audiograms 11, 12a and 12b).—Mr. A. A., aged 51, had had recurrent discharges from the left ear since childhood. The present illness began eight days before the first test of hearing was made. On Aug. 30, 1937 the ear showed a small perforation in the outer and upper segment of the left drum head, a small granuloma of the posterior meatal wall and a moderate amount of purulent discharge. On September 1 the secretion and the inflammatory reaction of the drum head were considerably reduced. On September 13 the discharge had been absent about five days and the perforation was healed. The drum head was still congested. No signs of congestion were present on September 27, when the last test was made. The right ear had had a normal appearance during the whole period of observation.

QUESTION OF OBSERVATIONAL ERRORS

Before attempting any explanation of these numerous variations in bone conduction one should determine whether they were not caused by variations in the technic of the tests or by errors in observation. For that purpose the bone conduction curves obtained from the noninflamed contralateral ears may be studied.

The contralateral tests for those recorded in audiogram 6b of chart 2 are recorded in audiogram 5 of chart 1. The latter audiogram relates to the right ear. This ear never showed any signs of congestion, while the left ear had severe suppuration. The right ear was tested only twice, for bone conduction, on January 8 and on June 30, 1937. It is interesting to note that the curve for the noninflamed right ear showed a see-saw motion like that of the curve for the inflamed left ear, but not so marked. There were 9 other patients in this series whose noninflamed ears gave bone conduction curves with a small see-saw motion. The reason for this interesting phenomenon is well seen in audiogram 5a of chart 2. This is a record of bone conduction of both ears of the patient in case 3, as registered two years later, when the left ear was again inflamed and discharging. The curves taken by the conventional method of testing for bone conduction are compared with the curves obtained while the opposite ear was masked. All curves are of the same date. The masking was performed by 60 decibels of a combined noise produced by a buzzer and an air current in an apparatus which I have described elsewhere.[3] It is evident from this audiogram that testing the bone

3. Hůlka, J. H.: A Masker for Bone Conduction Tests, Ann. Otol., Rhin. & Laryng. **47**:153 (March) 1938.

conduction without masking gave two partially false curves: the graph for the low tones of the healthy right ear was too high, and the graph for the high tones of the diseased left ear was also too high. A special inquiry was made during the test without masking as to whether the patient heard the threshold in the tested or in the contralateral ear. It was found that the threshold was localized in the tested ear, though the intensities above threshold were sometimes localized in the contralateral ear. Similar results were obtained in masking tests of 5 other patients of this series. One of these patients, however, lateralized even the thresholds in that contralateral ear which had the better hearing under the masking test in the respective frequencies. It should be stated here that the decrease in hearing of the low tones in the right-sided masking test in audiogram 5a could not have been caused by the fatigue resulting from the masking noise, because the left ear was masked first.

Audiogram 5a can be explained by the hypothesis that the record of the bone conduction test of either ear when taken without masking was the result of stimulation of both cochleas. This hypothesis is supported by my experience that in total unilateral deafness no linear decrease of bone conduction was observed in the good ear when the 60 decibels of masking noise was applied over the deaf ear. Several such curves are shown in the publication just cited, and a few more are in my files.

This interaction of the cochleas in the bone conduction tests without masking should be kept in mind as one examines audiograms 11 and 12b of chart 2. Using curves 4 (taken when the left ear was healed) for reference, one sees that: (*a*) With the increase of hearing of the low tones in the acute stage of the left ear that of the low tones of the right ear was also elevated (curves 1). (*b*) With the decrease of hearing of the low tones during the healing stage of the left ear that of the low tones of the right ear was also depressed (curves 3). (*c*) The hearing pattern of the right ear remained essentially unchanged. These facts suggest that: (*a*) The patient was a good observer, for the records of distracted observers on subsequent examinations show erratic patterns. (*b*) The technic of the test (for instance, the application of the receiver against different points of the mastoid process in subsequent tests) was not responsible for the movement of the curves, for then illogical or paradoxic curves would have resulted.

Such a paradoxic record is curve 3 of audiogram 9 in chart 2. One may see on audiogram 10b, which is the bone conduction record of the inflamed left ear of the patient in case 5, that curve 3 is substantially below curve 4. Contrary to expectation, in the record for the contralateral noninflamed ear curve 3 is above curve 4. This paradoxic position, however, was not caused by variation in the technic of testing. Though the right ear did not present any visible inflammatory changes,

the tragus curves and the air conduction curves—which are not shown in this essay—presented variations characteristic for Ménière's syndrome. The ear had its own movement of the bone conduction curves.

A description of those curves would lead into a different field. Of interest here is the exactness with which the pattern of the hearing on the right side was preserved while the low tones of the hearing on the left side showed considerable variation. It would be illogical to assume that the observations on the right ear were constantly exact, while those on the left ear varied with the technic or with the attention of the patient.

To a similar scrutiny were subjected all records on patients who were tested three or more times during the period of the disease. A study of 18 such patients in this group and of numerous others did not justify admission of observational or technical variations amounting to more than 5 (rarely 10) sensation units and that in only one to three frequencies of one curve.

EXPLANATION OF THE ATYPICAL EARS

Study of the atypical movements in the typical ears suggests that in the "atypical" ears of table 5 a certain stage or stages were missed either by the tests or by the course of the pathologic process. Each of the 4 ears of types I and II (table 5) was tested only once, the comparison being made with the contralateral, noninflamed ear. Here evidently the typical see-saw movement was missed because of an insufficient number of tests. On the ear of type III two tests were performed, one during the acute inflammatory and suppurative stage, the other after healing. The patient, however, did not come for examination until two weeks after the discharge appeared and the period of increased bone conduction could have appeared at the beginning of the inflammation. Of the 3 ears of type III, 1 was tested three times and 2 four times. Two of the discharging ears had granuloma, and in each case there was a long history of continuous discharge with intermittent irritation. The third was tested for the first time eighteen days after the beginning of the acute symptoms. In all 3 there was continuous improvement of the hearing of both the high and the low tones during the course of the treatment. One can conclude that these ears were probably not tested during the stage when the bone conduction for low tones was increased.

COMPOSITE PICTURE OF THE BONE CONDUCTION MOVEMENTS

Turning attention to the ears which were observed early in the acute stage of an inflammatory process in the middle ear and on which frequent

observations were made, one can outline the following picture of the bone conduction movements during and after the acute stage:

1. (*a*) During the acute stage there is an increase in hearing of low tones through the bone. This was observed in 33 of 38 ears studied.
   (*b*) At the same time there is a decrease in hearing of high tones by bone conduction. This was observed in 29 of 38 ears.
   (*c*) The actual depression of hearing of high tones may be greater than appears on the audiogram made by the conventional method, because the loss may be masked either by the interference from the contralateral, noninflamed ear (audiogram 5a of chart 2) or by a linear increase of bone conduction (curve 2 of audiogram 6b and curve 1 of audiogram 10b, both of chart 2).
2. (*a*) As the inflammation lessens, there is a gradual decrease of the elevated low tone hearing (curve 2 of audiogram 12b).
   (*b*) The hearing of high tones during the same period may be more depressed (curve 2 of audiogram 12b) or may not change or may show recovery.
3. (*a*) Before all inflammatory reaction disappears there may be a period when the hearing of low tones is depressed below the reference curve obtained by a test on the completely healed ear (curves 3 of audiogram 6b and 10b).
   (*b*) In this period the hearing of high tones already shows improvement (curves 3 of audiograms 6b and 10b).

One sees that the typical see-saw movements of chart 1 are not attained by a simple process similar to the action of a lever, with force applied at one end and weight at the other, with a fulcrum in between. The procedure is more complicated, suggesting action of several factors.

THEORY OF THE INCREASE IN HEARING OF LOW TONES

The inflammatory increase of bone conduction in the low frequency ranges is well known, and several theories have been advanced to explain this phenomenon. Bezold (quoted from Kerrison [4]) interpreted it as follows:

> 1. That normally the whole conducting chain—drum membrane, ossicles, and annular ligament connecting the foot-plate of the stapes to the rim of the oval window—is in a state of perfectly mobile equilibrium. 2. That any abnormal or pathological condition within the tympanum, either by changing the position of the stapes or inducing inflammatory changes in the region of the oval window,

4. Kerrison, P. D.: Diseases of the Ear, ed. 4, Philadelphia, J. B. Lippincott Company, 1930, p. 78.

places the fibres of the ligamentum annulare in some degree on the stretch. 3. That stretching of the annular ligament interferes with the transmission of sound waves reaching the ear by air conduction, but distinctly favors the propagation of sound waves through the cranial bones to the foot-plate of the stapes. According to this theory it is also obvious that ankylosis of the stapes due to hyperplasia of connective tissue about the oval window should also tend to increase and prolong hearing by bone conduction.

Bezold's theory will not hold its ground if one compares audiogram 12b with audiogram 10b. In the ear represented in 12b the Gellé test was positive through the whole course of the disease, while the ear represented in 10b showed fixation of the stapes, more so when the hearing of the small frequencies was decreased (curve 3). Among

TABLE 6.—*Results of the Gellé Test as Performed Thirty-Six Times on Twenty-Five Ears During the Acute Stage of an Inflammatory Process*

		Tests in Which Hearing of Ears Giving the Stated Reaction Was Increased or Decreased With				
		Low Tones		High Tones		
	Types of Gellé Reactions	Increase	Decrease	Decrease	Increase	Not Changed
+	(positive in all frequencies or negative in only 1)	8	4	8	2	2
i	(inverted in all frequencies).....	..	1	1	..	..
+i	(at least 1 frequency inverted, the rest positive)	6	1	6	..	1
+i−	(at least 1 frequency inverted, 2 or more positive, 2 or more negative	2	..	2	..	..
+−	(majority positive, at least 2 negative)	4	..	4	..	..
−+	(majority negative, at least 2 positive)	1	1	2	..	..
−	(all negative or only 1 +).....	7	1	6	2	..
	Number of tests made.....	28	8	29	4	3
	Number of ears tested.....	20	8	22	3	3

the 30 patients there were several types of Gellé reactions during the acute stage. They are shown in table 6.

Increase in hearing of low tones by bone conduction was associated in 28 tests performed on 20 ears with practically all varieties of the Gellé reaction. But also decrease of hearing of low tones was associated with all varieties of the Gellé reaction. In other words, there was no correlation between the increase or the decrease of hearing of low tones and the mobility of the stapes. In only 2 ears of the whole group did a totally negative Gellé reaction, associated originally with increase of hearing of low tones in the acute stage, change after healing into a totally positive Gellé reaction. One of them is ear 6 of charts 1 and 2. In the other ears either there was no change of the Gellé reaction after healing or the negative response appeared in a larger number of frequencies after the ear had healed. That would indicate that there was

less mobility of the stapes after cicatrization of the annular ligament than during the edematous softening of this tissue. The "inverted" reactions disappeared after the healing or their number diminished during the healing. They were replaced by positive reactions.

A similar lack of correlation was observed between the degree of increase of bone conduction and the Gellé reaction. For instance, the extremely high bone conduction curve 2 of audiogram 6b, taken on an ear in which the Gellé test was negative, could be matched with a curve of another ear, in which the Gellé test was positive, and so on.

The third hypothesis of Bezold finds no support in the light of these facts. Bezold's statement that "stretching of the annular ligament interferes with the transmission of sound-waves reaching the ear by air-conduction, but distinctly favors the propagation of sound waves through the cranial bones to the foot-plate of the stapes" is supported by some of the audiograms published here but is contradicted by others. So in the patient in case 1 (chart 1) the increased bone conduction of low tones is associated with decreased air conduction of the same frequencies in the right ear. But in the left ear air conduction did not drop with increase in bone conduction of low tones on Jan. 21, 1937. Air conduction curves 2 and 4 of audiogram 6a (chart 2) rose in the low tones with bone conduction curves 2 and 4 of audiogram 6b. In audiograms 12a and 12b bone conduction curves 1 and 3 show wide difference in the low tones, while the air conduction curves show considerable difference only in 512 double vibrations, with which frequency bone conduction shows the least variability. These examples are sufficient to cast a doubt on the absolute validity of Bezold's hypothesis.

Knudsen and Jones [5] explained increased bone conduction by the presence of noise in the testing room. An ear with unimpaired air conduction is disturbed by the noise and that disturbance impairs the acuity of hearing through the bone. On the other hand, when the air conduction is impaired by inflammation, the ear is not disturbed by the extraneous noise during the bone conduction test and the bone conduction curve appears elevated. While this observation is undoubtedly correct, it, again, does not explain the movements of the curves in the low tones in the bone conduction and the air conduction test of the patients in cases 3, 5 and 6, for some of the corresponding bone conduction and air conduction curves do not move in the opposite sense. Besides that, if the "increased" bone conduction of the inflamed ear is just a normal record on an ear deprived of the interference of noise, why should also the contralateral, noninflamed ear show better hearing? When only one test was taken, as in case 4, the Knudsen-Jones phenomenon was the most alluring explanation of the increased bone conduction.

5. Knudsen, V. O., and Jones, I. H.: Studies in Bone Conduction, Arch. Otolaryng. **13**:489 (April) 1931.

But of the 22 patients whose bilateral tests were taken two or more times, only 5 presented such relations of air conduction to bone conduction that the increased bone conduction could be explained by the extraneous noises masking the controls. The tests of 17 patients offered more numerous contradictions with increased number of observations. The masking effect of the noise should be admitted as a partial cause of the increased bone conduction in an acutely inflamed ear. But it is not the only or the whole cause of this.

Experiments of Hughson and Crowe [6] allow still another approach to the problem. Using the Weber and Bray effect, Hughson and Crowe found that the intensities of the sound impulses in the cochlea are increased if the round window membrane is immobilized by a periosteal transplant or by a cotton plug. They studied the function of the round window on air-conducted sounds in arbitrarily chosen frequencies between 250 and 8000 double vibrations. There is no reason to believe that a similar effect could not be observed on sounds conducted through the bone or on sounds of lower frequencies. It is quite reasonable to assume that purulent inflammation in the tympanum diminishes the elasticity of the secondary tympanic membrane by round cell infiltration, hyperemia, swelling or deposit of secretion.

THEORY OF THE DECREASE IN HEARING OF HIGH TONE

If one wants to accept the blocking of the round window as an explanation of the increase in bone conduction during the acute inflammatory stage, one has yet to explain a substantial difference between the findings of Hughson and Crowe and the observations described in this essay. Table 5 shows that in 32 of 38 ears the hearing of high tones during the acute stage was depressed, while Hughson and Crowe found that block of the round window caused elevation of the hearing of both the high and the low tones.

For the interpretation of this apparent contradiction the following hypothesis is advanced:

Hughson and Crowe published only experiments performed on ears free of infection, while in our group all ears were infected. The infection was on the promontory, i. e., on the outer wall of the basal turn of the cochlea. As demonstrated by Hughson and Crowe, Lurie and collaborators,[7] the basal part of the cochlea reacts to threshold intensities of the high tones. The blood supply of this region of the cochlea communicates with vessels of the bony capsule and these again are branches of the

6. Hughson, W., and Crowe, S. J.: Immobilization of the Round Window Membrane, Ann. Otol., Rhin. & Laryng. **41**:332 (June) 1932.

7. Lurie, M. H.; Davis, H., and Derbyshire, A. J.: The Electrical Activity of the Cochlea in Certain Pathologic Conditions, Ann. Otol., Rhin. & Laryng. **43**:321 (June) 1934.

regional arteries supplying the middle ear.[8] Therefore, the inflammation of the promontory can very well spread along the vessels and lymphatics as far as the spiral ligament and vascular stria of the basal turn of the cochlea. Atrophies of the vascular stria in the basal turn were found by Crowe and collaborators [9] in many ears with evidence of past inflammatory middle ear changes. In the acute stage of middle ear disease an inflammation of the spiral ligament and of the vascular stria could be responsible for the decrease of hearing of high tones by bone conduction. The depression of hearing of high tones appeared in human ears in which the inflammation was caused by infection, while in the ears of those experimental animals which were tested two to seven days after the operative procedure the hearing of high tones was increased, though some postoperative inflammatory reaction must have been present. That would indicate a toxic character of the impairment in the hearing of high tones. The toxins spread either through the cellular elements of the basal portion of the cochlea or, if they spread through the endolymph, their action was selective against the perhaps more delicate or more specialized cells reacting to the stimuli of high frequencies.

Observations on another group of ears of entirely different pathologic classification lead me to believe that there is still a third possible cause of an inflammatory decrease of bone conduction, namely, interference with the transfer of the acoustic vibrations from the bony capsule to the acoustic receptors proper along and through the spiral ligament, vascular stria and basilar membrane. In that group of ears decreased bone conduction was observed while in the same frequencies and at the same time an unimpaired response was obtained to stimuli propagating through the membranous cochlea across the annular ligament from the tragus. This third hypothesis would explain also the lack of depression of hearing of high tones by air conduction in the experimental animals.

Before accepting the cochlear explanation of the decrease in hearing of high tones, one should inquire whether the same effect could not have been caused by inflammatory changes in the transmission apparatus or in the round window or by some technical conditions of the tests.

Table 6 reveals that decrease in hearing of high tones occurred in ears in which the Gellé test was positive as well as in ears in which this test was negative. In the latter group the function of the ossicular chain was eliminated by the rigidity of the annular ligament. Therefore, the depression of the hearing of high tones must have occurred independently of the transmission mechanism.

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8. Shambaugh, G. E.: Communication Between the Blood-Vessels in the Membranous Labyrinth and the Endosteum and Those in the Bony Capsule of the Labyrinth, Arch. Otol. **34**:496, 1905.

9. Crowe, S. J.; Guild, S. R., and Polvogt, L. M.: Observations on the Pathology of High-Tone Deafness, Bull. Johns Hopkins Hosp. **54**:315 (May) 1934.

Fenestra rotunda is often mentioned as a direct pathway of the high tones into the basal turn of the cochlea, and it might be argued that the depression of the high tones was a direct result of the block in that region. But if applied for the threshold intensities this hypothesis is contradicted by the experiments of Hughson and Crowe in which the block of the round window did not raise the threshold of the high tones.

Another cause of the movements of the curve in the high tones could be the escape of the high tones from the bone conduction receiver. An ear with good air conduction might hear the high frequencies through the air and not through the bone. In order to eliminate this error, I check all bone conduction thresholds above 1012 double vibrations by removing the receiver a few millimeters from contact with the mastoid integument. In none of the tests of this group did escape of high tones from the receiver interfere with the recorded bone conduction threshold.

Finally, one again has here the phenomenon of the hearing in the healthy contralateral ear following through depression and recovery the hearing of high tones in the diseased ear. That decidedly suggests a cochlear origin of the high tone movements.

Recapitulating, the theory of movements of the bone conduction curve in acute purulent and also sometimes in acute nonpurulent otitis media is as follows: In the acute inflammatory stage the secondary tympanic membrane—and sometimes also the stapedial ligament—become rigid. This prevents escape from the cochlea of a certain fraction of that part of the acoustic energy which propagates to the organ of Corti from the bony capsule across the perilymph. The result is that a smaller output from the bone conduction receiver suffices for elicitation of threshold sensation in all frequencies; i. e., the bone conduction appears increased. In the high tones, however, this effect is more than counterbalanced by inflammatory changes which have reached the spiral ligament from the adjacent promontory and produced either a local toxic cellular or a diffuse toxic selective or combined intracochlear lesion. Thus the elevation of the bone conduction curve appears only in the low tones. After healing of the inflammation, both the effect on the round window and that on the basal turn disappear, and the typical see-saw movement of the curve results.

This theory easily explains also all the atypical movements of the curve. Either block of the round window or the inflammatory effect in the basal turn may occasionally be absent, especially in recidives of old processes. Analogically, the decrease in conduction of the low tones observed in some ears during the healing stage can be interpreted as due to temporary edematous softening of the secondary tympanic membrane followed by normal or even increased rigidity after complete healing.

The effect of tympanic inflammation on the round window and that on the basal turn are two independent factors with independent variations of activity. Though dominating, they are not the only factors determining the movement or the position of the bone conduction curve of a given ear. The other factors are (*a*) the interference of the contralateral ear, (*b*) the amount of damage done to the ear by previous processes and (*c*) the interference of the meatotympanic component of the bone conduction test, i. e., the interference of the transmission system in the bone conduction test. Remembering this and keeping in mind also that there is no strict localization of definite frequencies in the cochlea, though in moderate and in threshold intensities the base reacts to high tones and the apex to low tones, one may understand why there

TABLE 7.—*Fulcrum of the See-Saw Movement*

Location of the Fulcrum	Ears Inflamed	Ears Contralateral, Noninflamed
512–1024	8	1
1024	6	1
512–2048	5	..
2048	3	1
256–2048	3	..
256–512	2	..
512	1	2
256–1024	1	2
256	..	1
128–256	..	1
	29	9

is no strict pitch limit in table 7 in terms of the location of the "fulcrum" around which the see-saw movement occurred. When the "fulcrum" spread over several frequencies of the audiometer, the variation in that region did not exceed 10 sensation units. This sparing of the middle tones was observed only in bone conduction and not in air conduction, the latter being characterized by linear movements of the threshold curves.

SUMMARY

Changes in bone conduction caused by acute inflammation of the middle ear were studied audiometrically on 38 inflamed and 22 noninflamed ears of 30 patients. Hearing during the acute stage was compared with hearing after the ears were healed. It was found that the tympanic inflammation increased the bone conduction of low tones and simultaneously decreased the bone conduction of high tones in 29 of the 38 inflamed ears. On the graph this change had the appearance of a see-saw movement of the bone conduction curve. Besides this typical

movement, several kinds of atypical shifts of bone conduction curves were observed. Neither the Bezold theory of a stretching of the stapedial ligament nor the Knudsen-Jones theory of a masking by noise could explain these phenomena. In this essay the following explanation is offered:

1. Acute inflammation in the tympanum blocks the round window.
2. The block causes increased bone conduction in the whole range of frequencies.
3. This general increase is more than counterbalanced in the high tones by the penetration of toxins through the promontory into the basal turn of the cochlea. That causes depression of the conduction of high tones, while the conduction of the low tones remains increased.
4. Either the effect of the tympanic inflammation on the round window or that on the basal turn may be absent in some ears or in certain stages of the inflammation. This accounts for the atypical movements of the bone conduction curves.

Conversely, by observing the bone conduction movements in acute otitis media one can reach a clinical evaluation of the progress and extent of the aural lesions.

SPHINCTER ACTION OF THE LARYNX

JOEL J. PRESSMAN, M.D.
LOS ANGELES

The larynx is looked on primarily as a sound-producing organ—a structure capable of producing sound waves to be converted by other structures into speech. Practically all physiologic research on the larynx is directed toward these mechanics, and a vast store of knowledge concerning the function of the structure in speech has been accumulated. Obviously, the importance of such a function is not to be minimized; nevertheless, this one-sided view has distorted the proper concept of laryngeal physiology. It has relegated to the background those physiologic activities of vast importance which represent not only the stimulating factor in the early evolutionary development of the larynx but the principal reason for the persistence of this organ through the phylogenetic scale and for its presence throughout the mammalian kingdom.

Like all other mammals, man has a larynx developed not primarily for purposes of speech but rather to serve as a sphincter valve, chiefly to prevent the invasion of anything but air into the lower respiratory passages but serving other sphincteric purposes as well, the mechanism being called into play whenever it becomes necessary that the lower respiratory passages be functionally separated from the upper. To this, the development of a sound-producing function within the larynx is decidedly secondary and almost accidental. If speech were not part of human physiology, a larynx, perhaps not in its present form, would nevertheless exist. Such is the case in a great variety of present day species which produce no sound, as, for example, the bear. This concept of a primary sphincteric function is, as Negus [1] pointed out, by no means new but was described in 1867 by Marshall and again in 1892 by Stuart.

The need for a larynx and its beginnings as a sphincter valve rather than as a sound producer can best be comprehended by a study of its

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The photographic studies which constitute part of this work have been made in association with Mr. Arthur Hinman. Pressman, J. J., and Hinman, A.: A Simple Technique for Taking Motion Pictures of the Larynx in Action, Arch. Otolaryng. **26**:526-530 (Nov.) 1937; Further Advances in the Technique of Laryngeal Photography, Laryngoscope **50**:535-546 (June) 1940.

1. Negus, V. E.: The Mechanism of the Larynx, St. Louis, C. V. Mosby Company, 1929.

evolutionary development. A thorough, masterful treatment of this subject can be found in the early pages of Negus' text, already cited. Sketchily, the situation can be summarized by pointing out that the most primitive larynx makes its appearance in the lungfish. Before this in the evolutionary ladder no need existed for a valve to protect lower respiratory passages, since there were none, absorption of oxygen taking place from the water through the medium of gills. However, with the development of a lung bud and ultimately of a lung connected by a windpipe to the pharynx, as in lungfish, a new situation arose. Here an air-breathing apparatus developed in an organism primarily adapted for living within the sea. This arrangement, however, could not be maintained without some mechanism for physiologically isolating the lung from continuity with the pharynx, because the increased pressure of the water, as compared with air pressure within the lung, would cause sea water to flow through the trachea and enter the lung whenever the lungfish opened its mouth. Thus, this species could not exist as such, for its lungs would be destroyed or at least rendered functionless almost at the moment of its birth. To obviate this chain of events there appeared within the structure of the upper end of the trachea a simple circular group of muscle fibers, constituting an encircling sphincteric band. When the muscle band closed, the lower respiratory passages were effectively isolated from the upper. The fish could now, with the sphincter closed, open its mouth and swallow food without jeopardizing the newly developed lungs, since it was quite impossible for the water in which it swam to pass the constricted sphincter band. In these simple physical and physiologic phenomena are found, first, the need for a larynx, and, second, evolution's preliminary answer to this need. Not by any stretch of imagination can this simple, most primitive larynx be pictured as having been developed for purposes of phonation, yet by chance even it was capable of sound production. Such capabilities can be interpreted only as an accidental by-product to the need for a sphincter. That such a larynx in which a sound-producing function was never intended can, in spite of this, actually produce sound is evidenced in everyday life through such a comparable physical example as the production of sound by the escape of air from a football bladder, especially if the lumen of the neck of the bladder is further constricted by the fingers. In this comparison the football bladder simulates the lung and the narrowed neck of the bladder represents the sphincter narrowing the trachea. Another example of sound produced by the escape of air under pressure from a large chamber through a narrowed orifice is that resulting from the escape of air from the colon through the anal sphincter.

Even in primitive states, then, in which there is no need for laryngeal sound, potentialities for its production are found. That ultimately such a phenomenon serves a useful purpose in no way changes the proper concept of the larynx as an organ the sphincteric function of which is far more important in bodily economy than its secondary use as a provider of tones.

Through an evolutionary cycle characterized in part by the development of forelimbs, as first seen in reptiles and later in mammals, including man, this heritage of a sphincter girdle about the trachea persisted in each succeeding species throughout the evolutionary scale. Gradual changes have taken place in its anatomic structure, but it nevertheless has stubbornly maintained its essential quality of a circular constricting band, the functioning of which is known as "the sphincter action of the larynx."

In keeping with the progressive development of the species, the sphincter mechanism came to serve additional useful purposes beyond that of protecting the lower respiratory passages during swallowing. As the result of certain modifications in anatomic structure, the larynx in man is far more complex than the original group of circular fibers which surrounded the upper respiratory tube as exemplified in the lungfish. Extensive evolutionary changes have taken place. First, a rigid protective cartilaginous framework appeared, in order that the larynx might, despite the prolonged presence in the esophagus of a huge bolus of food, retain a patent lumen. Next, the division of this framework into several cartilages occurred as well as the division of the original single circular muscle into various groups of muscles, each serving its own purpose. Yet, despite these changes the primitive sphincter girdle is still recognizable even in the most highly developed and complex larynx.

During the course of evolution the sphincter girdle ultimately took the form of paired horizontal muscular bands, one on each side of the interior of the cartilaginous framework, with no division into true or false cords. In this form it is still present in many mammals, such as the ox. In man, however, as well as in certain other mammals, these sphincteric bands divided themselves by a horizontal fissure, known as the ventricle of the larynx, into two portions, one above the other. The lower of these are recognizable as the true vocal cords, or vocal folds, and are used chiefly for sound production. The upper are the false vocal cords, or ventricular folds, which form the chief but not the sole elements of the sphincter mechanism. It is not to be presumed, however, that these false cords serve only in the sphincteric processes. They play a certain minor role in the phonatory mechanism as well, but this phase of their activity is not part of the present consideration and therefore will not be discussed further. It is well that in man such a division into true and false cords took place. Had

a single band been retained, it would of necessity, as is the case in the ox, have been too gross and crude a structure to produce a pleasant, variable voice; or if it had developed as a single delicate band capable of producing fine tones, such delicacy of structure would in all probability have rendered it ineffective as a powerful sphincter. It is extremely interesting and important to note that the relative importance of the sound-producing and sphincteric mechanisms is hinted at in nature by the fact that in species in which the larynx is designed primarily for only one purpose the anatomic structure of the organ is usually better adapted to the sphincter action than to the production of sound. In other words, in a great variety of species there is found a single band capable of highly effective sphincteric activities but producing only crude ineffective phonatory results, whereas the reverse is seldom true.

Whenever the need for a highly specialized voice warrants the presence of vocal bands designed primarily for the production of sound, these have developed as an adjunct to, but almost never to the complete exclusion of, the sphincteric apparatus. Thus, in man the true cords are more or less specialized for the purpose of voice production, and the sphincteric organ is effectively present in the form of the false vocal cords and allied structures.

ANATOMY OF THE FALSE CORDS AS A LARYNGEAL SPHINCTER GIRDLE

The false vocal cords are the principal structures involved in the sphincteric action. Essentially these are two thick folds situated above the level of the true vocal cords and separated from them by the ventricle. They are composed chiefly of striped muscle tissue covered with mucosa but include a narrow band of fibrous tissue known as the ventricular ligament.

The rather complex thyroarytenoid muscle (fig. 1 *A*) arises anteriorly from the lower half of the angle of the thyroid cartilage and from the conus elasticus (or cricothyroid membrane). It almost immediately divides into two portions. The deeper and more internal portion of muscle passes backward below the level of the ventricle to become an intrinsic part of the true vocal cord. It is not this portion which is of interest at the moment.

The second portion, passing on a higher plane, is the superior external division. It arises from the common point of origin already mentioned, from which it passes backward along the inner surface of the thyroid cartilage. Some of its fibers constitute the outer wall of the ventricle of the larynx. Its chief mass, inserting on the front face of the corresponding arytenoid cartilage, forms the bulk of the false vocal cord and therefore constitutes the principal element in the

formation of the sphincter girdle. Some few of its fibers, however, do not terminate at the arytenoid cartilage but pass around its lateral aspect to join the interarytenoid muscle, blending intimately with this structure (fig. 1*B*).

Thus, if the right and left muscles are considered as a single unit, together with the interarytenoid muscle, with which their fibers blend, there is recognizable a complete circle of muscle surrounding the

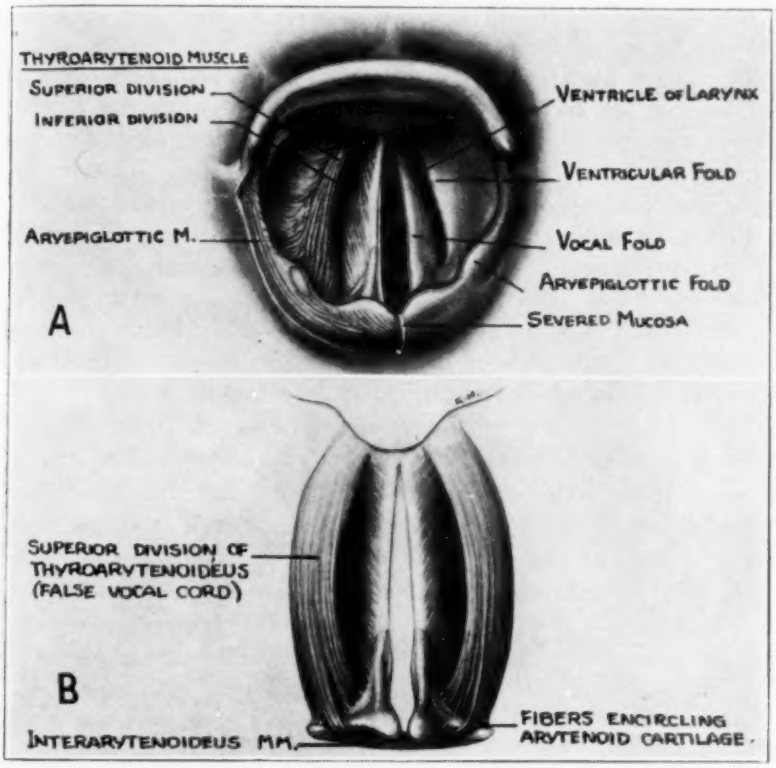


Fig. 1.—*A*, the division of the thyroarytenoid muscle into superior and inferior portions to form the false and the true vocal cords, respectively. The mucosa is intact on the right. *B*, the passage of fibers of the superior division of the thyroarytenoid muscles around the arytenoids to join the interarytenoid muscles in the formation of the "arytenoid sling." Fibers similarly pass anteriorly from the interarytenoid muscles to enter the false vocal cord from behind.

interior of the larynx. This encircling muscle band can conveniently be termed the "arytenoid sling," and it is this sling which phylogenetically represents the sphincter band of the lungfish. The circular origin and circular distribution of this group of muscles account for the "purse string" or "puckering" action when the sphincter closes.

MODE OF ACTION OF FALSE CORD SPHINCTER

All the details of the interplay of muscular activity by means of which this sphincteric action becomes possible are not well known. Therefore, much of what follows is conjecture, although I think one can be reasonably certain that the general principles involved are for the most part correct.

It is apparent that some muscular mechanism is present to control the opening and closure of the false cords independently of movements of the arytenoids even though the muscles of the false cords insert into the bodies of these cartilages; otherwise the false cords would exactly follow movements of the true cords in abduction and adduction, so that the former would completely close when the true cords were completely closed, as, for example, during phonation. This obviously does not take place. It must be conceded, however, that a slight degree of approximation of the false cords toward the midline in all likelihood occurs as an accompaniment of similar movements in the true cords, since both structures insert on the arytenoids and must rotate with them. But such movement of the false cords toward the midline as part of rotation of the arytenoids is not great, is limited to the posterior extremities of the cords and in no way approaches the degree of closure which can ultimately be accomplished.

The closing action of the sphincter begins by internal rotation of the arytenoids. The latter movement is accomplished by the action of those adductor muscles which arise anteriorly and insert on the muscular process of the arytenoids. These are (1) the lateral cricoarytenoid muscles and (2) those fibers of the thyroarytenoid muscles (both superior external and inferior internal divisions) which insert on the muscular process. In addition, the arytenoids are pulled together toward the midline by the approximating action of the interarytenoid muscles. This closes the true cords, preliminary to the sphincteric closure, and approximates the posterior extremities of the false cords.

This preliminary phase of closure having been completed by the internal rotation of the arytenoids, the actual sphincteric closure begins. The latter results from the puckering action of the following groups of fibers which, as I have pointed out, constitute the arytenoid sling:

1. Fibers of the superior division of the thyroarytenoid muscles arising anteriorly in the wedge of the thyroid cartilage and terminating at various points in the false cord itself, possibly in the ventricular ligament.
2. Fibers (apparently also a portion of the superior division of the thyroarytenoid muscles) which arise more laterally in the

conus elasticus and curve upward and backward, following a shorter course directly into the false cords.

3. A third group of fibers of the superior thyroarytenoid muscles, which do not terminate in the false cords but continue around the lateral aspect of the arytenoid to join the interarytenoid muscles.
4. Fibers from the decussation of the interarytenoid muscles which curve forward around the arytenoid cartilages to enter the false cords from behind.

The puckering or sphincteric closure which can be accomplished by the action of this group of muscles most likely could not be accomplished satisfactorily without some foreshortening of the false cords as well as foreshortening of the true cords. From direct observations made during the performance of this closure and from photographic measurements, it seems as though the entire larynx becomes shorter in its anteroposterior diameter during the closure of the sphincter.

Assuming that such foreshortening actually takes place, it would be logical to believe that it results from a generally backward movement of the thyroid cartilage in conjunction with a forward movement of the cricoid cartilage, the latter carrying the arytenoids with it. (This is independent of the elevation of the larynx in the neck, which takes place from the action of the extrinsic muscles.) This approximating movement of the two cartilages would have the effect of bringing the posterior limits of the larynx closer to the anterior and would result in a shortening and flaccidity of both the true and the false cords, not only enabling the puckering sphincteric movement to take place more effectively but, in addition, providing the relaxation necessary for the forward tilting of the arytenoids to fill the posterior gap, as will be described later. The backward displacement of the thyroid cartilage is made possible by the relaxation of the cricothyroid muscles (fig. 2*A*), while the forward movement of the cricoid and the arytenoid cartilages is made possible by a relaxation of the inferior constrictor of the pharynx (fig. 2*B*). With these two muscles relaxed, foreshortening may be accomplished by the forward pull on the arytenoids of the inferior and the superior divisions of the thyroarytenoid and the lateral cricoarytenoid muscles and their backward pull on the thyroid cartilage.

It has been assumed that the cricoid cartilage is moved forward as an indirect result of the anterior pull exerted not directly on the cricoid but on the arytenoids, which, of course, articulate with the cricoid. If this does occur (and it is not at all certain but that the forward movement is limited solely to the arytenoids), it becomes necessary to assume a certain degree of fixation between the base of the arytenoid

and its facet on the cricoid. It can be imagined that such fixation takes place as a result of the action of the cricoarytenoid ligaments and of contractile efforts of the posterior cricoarytenoid muscles, which arise on the posterior face of the cricoid and insert on the posterior surface of the muscular process of the arytenoid. Assuredly more

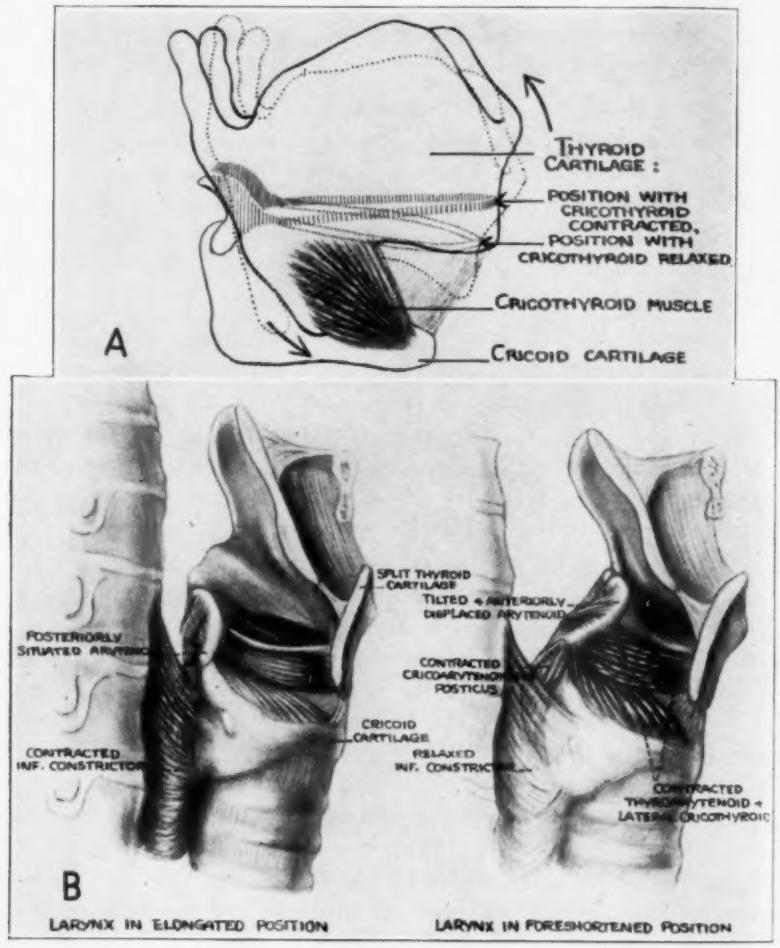


Fig. 2.—*A*, schematic drawing to illustrate that contraction of the cricothyroid muscles lengthens the interlaryngeal diameter and that their relaxation contributes to foreshortening. *B*, when relaxation of the inferior constrictor of the pharynx takes place, the forward pull of the anterior intrinsic muscles can effectively displace the cricoid and arytenoid cartilages in a forward direction. In conjunction with the action shown in *A*, this results in laryngeal foreshortening.

important than any movement of the cricoid itself is the forward tilting of the apexes of the arytenoid cartilages. This likewise is dependent on fixation and stability of the base of the arytenoids on their cricoidal

facets (fig. 3). This fixation and stabilization of the base need not prevent the anterior tilt of the arytenoids, an action which is known from direct examination and photographs to take place during the sphincteric closure.

As a result of this muscular play, especially the purse string action of the false cords, the sphincteric girdle closes tightly except that a posterior gap or dehiscence remains. This dehiscence is closed partially by the anterior displacement of the arytenoids already mentioned but chiefly by a tilting forward and inward of their apexes carrying with them the cartilages of Wrisberg and of Santorini. These structures thus come to lie over the most posterior portion of the sphincter, like an inset approaching it from above and behind. Most of this tilting action probably results from the constricting effect of the arytenoid sling, which passes behind the arytenoid cartilages. The fixation

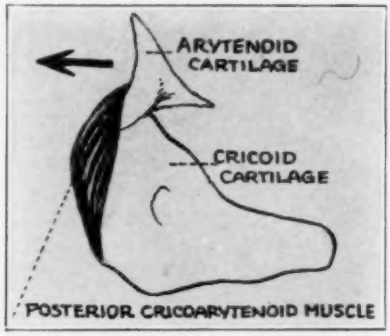


Fig. 3.—Contraction of the posterior cricoarytenoid muscle tends to stabilize the base of the arytenoid on its cricoidal facet.

of the base necessary for the tilting effect results from contraction of the posterior cricoarytenoid muscles, as previously mentioned.

THE ARYEPIGLOTTIC SPHINCTER

This, then, is the chief physiologic mechanism involved in the sphincteric closure of the larynx. The sphincter described is not, however, the only one present. Another, possibly as efficient, is located on a plane superior to it. The chief anatomic structures involved are the aryepiglottic folds. These do not phylogenetically represent the primitive sphincter girdle, since they are not present in the more primitive larynxes. It is not essential that this sphincteric valve and the other, composed of the false cords, function simultaneously, although this often occurs. In fact, surgical or pathologic destruction of one valve or the other passes practically unnoticed from the subjective viewpoint. Thus, it is possible to amputate the epiglottis and render

the aryepiglottic sphincter inefficient, in which case the action of the false cord sphincter alone proves entirely adequate and no inconvenience is suffered by the patient. On the other hand, when a false vocal cord (or even both false cords) is surgically removed, as in the more extensive laryngofissures, the aryepiglottic valve serves efficiently to provide a sphincter mechanism that functions almost as well as the two structures combined.

This second sphincter valve is formed chiefly, as I have mentioned, by the action of the aryepiglottic folds (fig. 4 *A*). These are broad

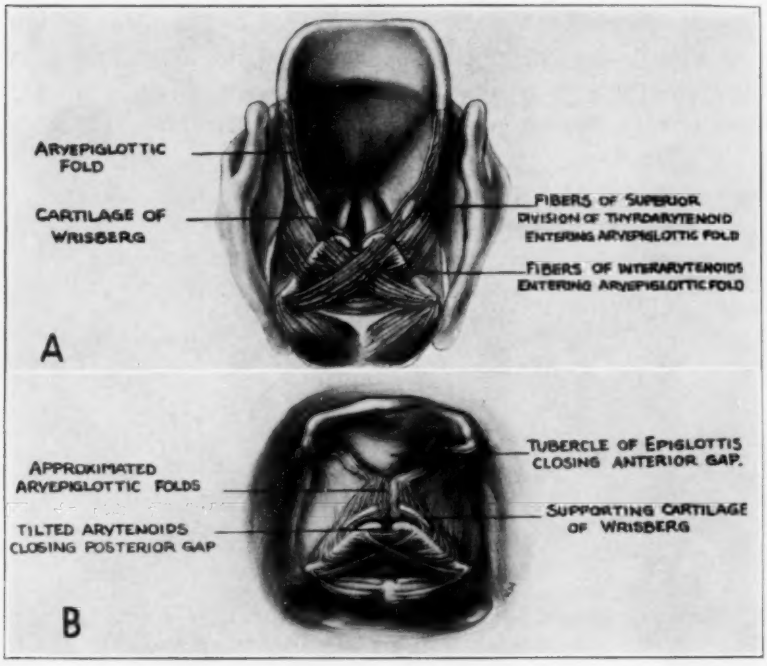

ARYEPIGLOTTIC FOLD
CARTILAGE OF WRISBERG
FIBERS OF SUPERIOR DIVISION OF THYROARYTENOID ENTERING ARYEPIGLOTTIC FOLD
FIBERS OF INTERARYTENOIDS ENTERING ARYEPIGLOTTIC FOLD
A
APPROXIMATED ARYEPIGLOTTIC FOLDS
TILTED ARYTENOIDS CLOSING POSTERIOR GAP
TUBERCLE OF EPIGLOTTIS CLOSING ANTERIOR GAP.
SUPPORTING CARTILAGE OF WRISBERG
B

Fig. 4.—*A*, schematic dissection of the aryepiglottic folds. *B*, schematic dissection of the closed aryepiglottic sphincter.

musculomucosal folds, one of which forms each of the posterolateral boundaries of the larynx. As more or less vertical shelves, they pass from the arytenoid cartilage in an anterolateral direction, to terminate in the lateral margins of the epiglottis. Within each fold and near its posterior extremity is located the rodlike, vertically placed cartilage of Wrisberg (or cuneiform cartilage).

Within the confines of this aryepiglottic fold are found those fibers of the superior division of the thyroarytenoid muscles which curve upward to enter into the muscular structure of the fold. There are also to be found fibers of the interarytenoid muscles, which, curving

laterally around the arytenoid cartilage, enter the fold from behind and terminate either in the connective tissue of the fold itself or, extending far anteriorly, reach the epiglottis, inserting into the latter structure. These fibers are sometimes considered as a separate muscle, the aryepiglottideus.

When this sphincter mechanism is called into play, the contraction of the muscle fibers contained within the aryepiglottic folds approximates the latter structures one to the other, a certain rigidity being present because of the supporting factor provided by the strutlike sesamoid cartilages of Wrisberg (fig. 4 *B*), which are included in connective tissue compartments within the structure of the fold. The puckered aryepiglottic fold presents a posterior gap, which again is filled in by the mass of the arytenoids. As the result of an upward

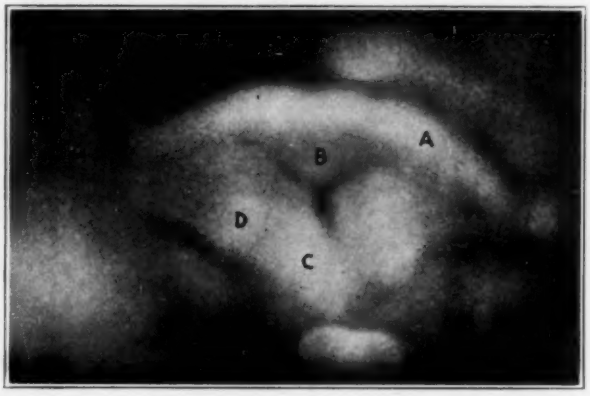


Fig. 5.—Photograph of the closed aryepiglottic fold (Bell Telephone Co.). *A* indicates the free margin of the epiglottis; *B*, the tubercle of the epiglottis; *C*, the prominence of the arytenoid cartilage, and *D*, the aryepiglottic fold.

movement in the neck brought about by the action of the extrinsic muscles and a lowering of the epiglottis by the pull of the aryepiglottic fold, the puckered sphincter contacts the tubercle of the epiglottis, which fills in any remaining anterior gap. This more superiorly placed tier of the sphincter mechanism, therefore, results from the apposition of the aryepiglottic folds laterally, the superimposition of the tubercle of the epiglottis anteriorly and the mass of the superior portion of the arytenoids posteriorly (fig. 5).

It is perhaps not entirely accurate to picture the aryepiglottic sphincter and the false cord sphincter as two distinct mechanisms, individually brought into action for one purpose or another. Essentially it is more likely a two-tiered mechanism often acting as a unit or at least with one part in close association with the other. However, under certain circumstances the false cords can act alone. The exact

role of each mechanism in sphincteric activities is not well known. From direct observation it would seem that the action of the aryepiglottic folds takes place chiefly when the sphincteric response is elicited by the stimulation of swallowing a large bolus of food and in physiologic acts such as gagging and vomiting. However, when the sphincter apparatus is used for thoracic fixation and during cough, as will be discussed later, it seems as though the false vocal cords play the chief role and the aryepiglottic structures a relatively minor part if any. Some approximation of the latter folds probably does take place, but their closure is not complete and apparently is physiologically ineffective. These observations, however, will possibly need revision as study of the problem progresses.

A third subglottic sphincter mechanism is sometimes mentioned, but if it exists it is not, strictly speaking, part of the laryngeal apparatus and need not be further discussed.

SEQUENCE OF EVENTS DURING CLOSURE OF THE FALSE CORD SPHINCTER

It is entirely possible to observe the appearance of the false cord sphincter as it undergoes the sequence of events leading to an airtight and water-tight valvelike closure completely isolating the lower from the upper respiratory passages. These movements can be studied most effectively in slow motion moving pictures, provided the exposure of the larynx for such photography is by direct laryngoscopic exposure, so that the epiglottis is retracted and out of the way. Occasionally the sphincter mechanism can be observed indirectly with the laryngeal mirror and possibly photographed in this way, but in the vast majority of cases direct exposure of the larynx with the laryngoscope is required. Under such circumstances one can watch and even record laryngeal activities during thoracic fixation, cough and swallowing, all of which are accompanied by the sphincteric closure. When studied by such methods as these, the sphincter valve action of the false cord is found to follow a definite plan, which does not vary regardless of the purpose it is intended to serve. The time element, however, varies considerably. When the sphincter action is part of a cough cycle, the opening and closing phases are extremely rapid and the period of closure is of very short, even momentary, duration. On the other hand, when the closure is utilized for thoracic fixation, these movements are relatively slow and the duration of the closure depends on the length of time thoracic fixation is maintained. Except for this factor of time, however, a description of the opening, closing and maintenance phases for any one purpose serves to describe the sphincter action at the false cord level under all circumstances.

Closure of the sphincter valve at this level in man consists not only of action by the false cords but is accompanied by closure of the true cords as well, although the latter probably does not contribute a great deal to sphincteric efficiency, especially when the purpose of the action is to trap air below the larynx. The closure begins by an approximation of the true vocal cords, beginning anteriorly. The anteriormost segments come together and close tightly, to be followed by a closure of greater and greater lengths in a progressive adductor movement which spreads from the anterior limits of the true cords in a posterior direction until the entire length of one true cord is in firm apposition with its fellow.

Finally, no hiatus is visible, the closure of the true cords being complete. This complete closure varies from that in phonation inasmuch as during the sphincteric action no hiatus is present anywhere

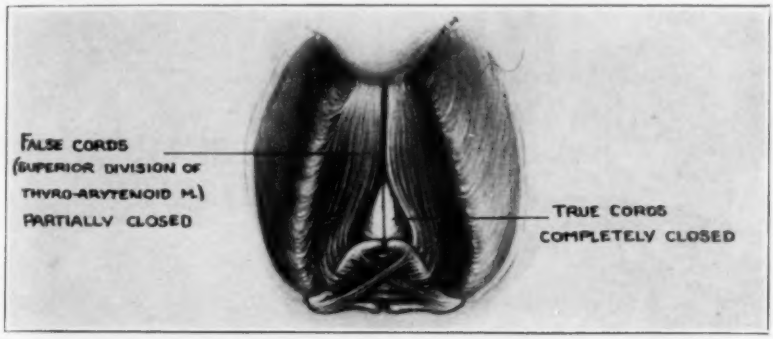


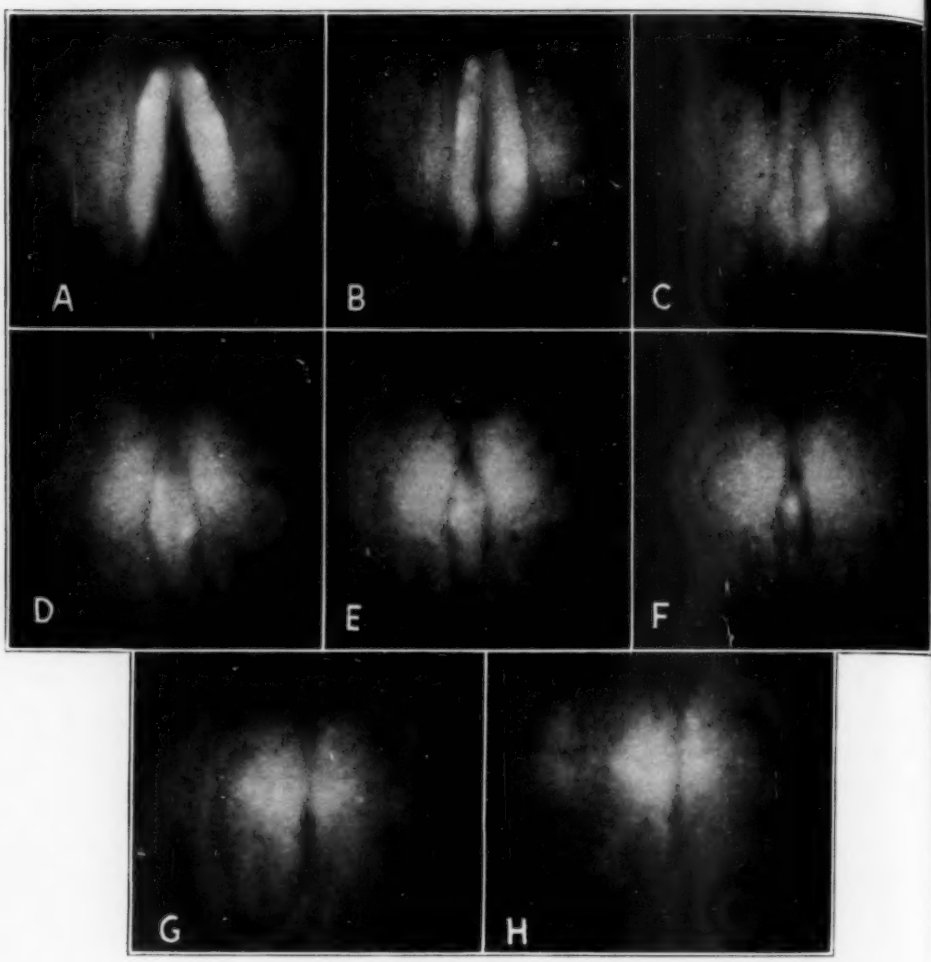
Fig. 6.—Diagrammatic dissection of the false cord sphincter in partially closed position. The short posterior segment described as closing early in the process is hidden by the arytenoids.

along the length of the cords, whereas in phonation a hiatus, even if very small, must invariably be present at some point between the true cords.

This preliminary closure of only the true cords, although not entirely effective, does possess some value as a sphincteric action, a point which will be further discussed in later paragraphs.

After complete approximation of the true cords there follows a similar closure of the false cords. The approximation of the latter structures begins at about the same instant the closure of the true cords is completed. Therefore, the true cords can be seen to close first, followed after a brief moment by closure of the false cords.

This movement of the false cords takes place in much the same way as that of the true cords. The closure begins anteriorly and proceeds posteriorly in progressive fashion, the false cords sweeping to the midline over the already approximated vocal bands. As this progres-



EXPLANATION OF FIGURE 7

Photographs demonstrating successive steps in the closure of the false cord sphincter valve.

A, preliminary stage: The false and the true cords are abducted in position of rest.

B, beginning of closure: The true cords are almost closed. The false cords are still widely open.

C and *D*, closure of the false cords begin: The anterior portions of the false cords begin to approximate, and there is also closure of their posterior extremities, leaving an oval hiatus. Note the complete closure of the true cords.

E, anterior segment of false cords completely closed.

F and *G*, continued closure of the false cords progressing from anterior to posterior.

H, final stage: The false cord sphincter is tightly closed, completely hiding the underlying true cords.

sion takes place, it can be seen that whatever portion has closed in any given stage of the cycle is closed very tightly, so that complete approximation of any given anterior segment takes place before the following posterior segments are in apposition. There is, however, an exception to the closure beginning first at the anterior limits of the false cords and progressing posteriorly. At the beginning of the action a very small length of the most posterior portion comes into contact with its fellow, so that the hiatus between the false cords is more or less oval. This is due to the action of the approximated arytenoids. With this exception, however, the closing action is a progressive one from the anterior to the posterior part (fig. 7).

When the false cords have been tightly approximated throughout their entire length, there is still a hiatus at the level of the posterior commissure. This gap is then sealed by a tilting forward of the arytenoids so that they come to fill the posterior gap somwhat in the fashion of an inset approaching it from above and behind. The appearance of the larynx at this state of complete closure gives the impression of a flat sheet of tissue completely occluding the lumen with a central longitudinal line of closure. The uppermost portion of the limb, extending about four-fifths the length of the larynx, lies between the margins of the false cords. The posterior limit of the vertical limb represents the line of closure between the arytenoid masses.

A visual study of the action of the arytenoids during the actual closing process is interesting. Apparently, at the beginning of the closure these rotate inwardly as far as possible, carrying the false and the true cords with them toward the midline. This inward rotation of the arytenoids results in complete apposition of the true cords and of a very small posterior segment of the false cords. From this point on, further closure of the false cords to the point of completion takes place of their own accord without additional internal rotation of the arytenoids. The former structures are therefore functioning as a true sphincter. When the false cords have been brought into apposition, a further squeezing or tightening is discernible, also entirely independent of all arytenoid movement and obviously occurring as a result of intrinsic muscular activity within the mass of the false cords themselves.

In the preliminary closure, which precedes that of the false cords, the true cords serve as a relatively adequate valve to prevent the ingress of air, fluids or solids into the trachea, but their closure is entirely inadequate to prevent the egress of air. The reason for this is shown on sagittal section: It is then seen that the true cords present superiorly a flattened, table-like surface, so that pressure from above is equally distributed over the entire surface of the approximated cords. Such equally distributed pressure exerts little effect in inverting the edges of the cords, so that despite pressure from above the barrier of the

approximated true cords remains intact. Thus the cords are relatively efficient as an inlet valve. In contradistinction to this, they are not at all efficient as an outlet valve, for the under surface of the approximated true cords is dome shaped (fig. 8), so that air pressure from below converges to the midline to be exerted in its full strength on the free margins of the approximated cords. The concentration of air pressure can with relative ease force the margins of the cords apart, creating a chink through which the pressure dissipates itself.

Fortunately, the sphincteric closure at this level is only in small measure dependent on the action of the true vocal cords, so that the sphincter mechanism proves adequate even when the true cords are

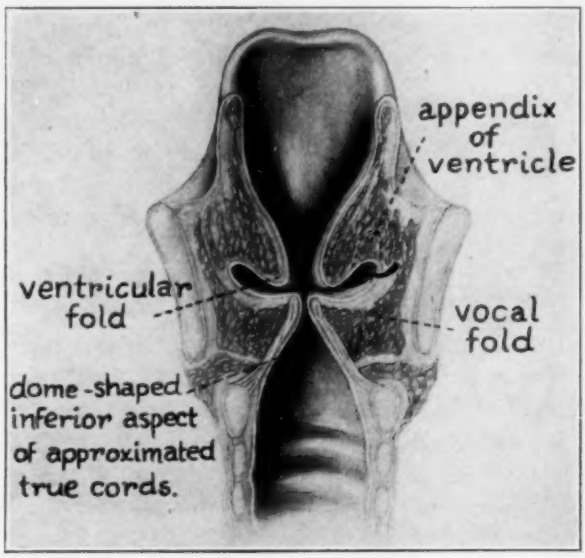
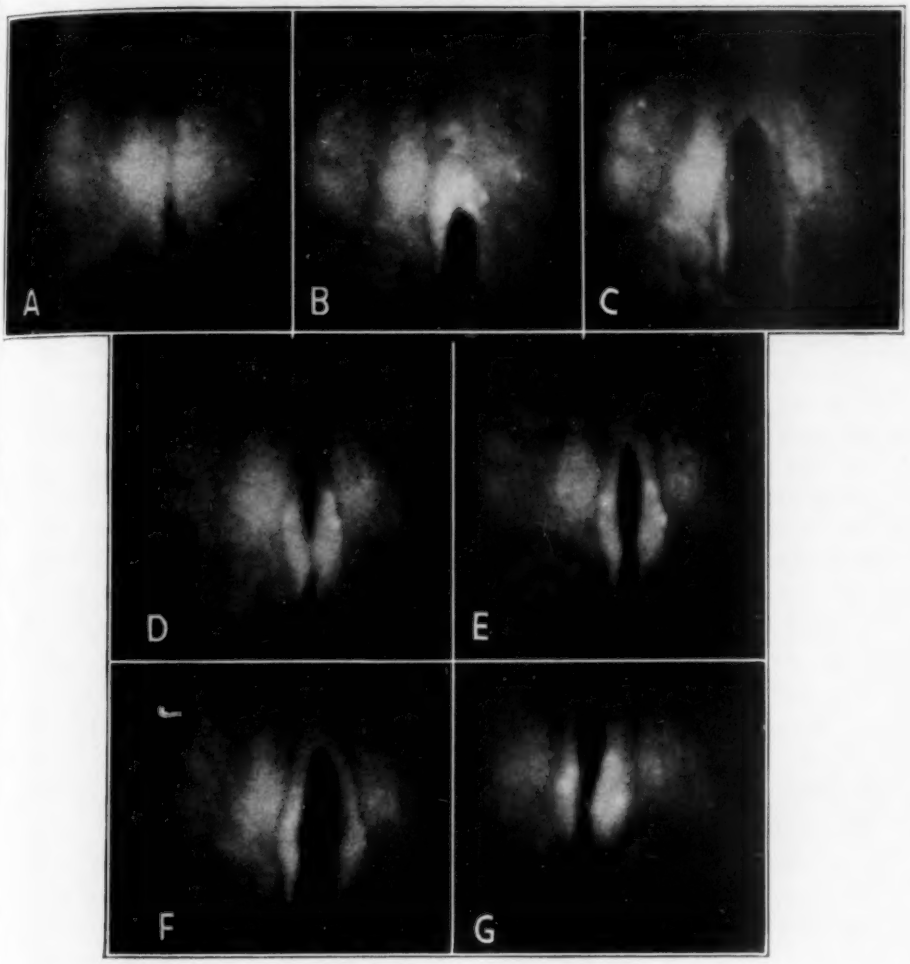


Fig. 8.—Sagittal section of the larynx, demonstrating the dome-shaped inferior surface presented by the closed true cords.

surgically removed. By far the greater effect comes from the closure of the false cords.

RELEASE OF THE SPHINCTER MECHANISM

When sphincteric closure has been maintained intact for as long a period as is necessary, its release is characterized by sudden relaxation of the sphincter girdle. The true and the false cords fly open almost as a unit. This is brought about or at least accompanied by an outward rotation and lateral separation of the arytenoids and a general relaxation of the internal structures of the larynx. Both the true and the false cords, but especially the former, have the appearance of a considerable degree of flaccidity. If the purpose of the sphincteric closure



EXPLANATION OF FIGURE 9

Photographs demonstrating successive steps in the release of the false cord sphincter (purposeless waving and repeated irregular openings and closings of the true cords).

A, the false cords are adducted, the sphincter closed.

B, the false cords are open throughout their entire length. True cords begin to open posteriorly. The arytenoids are rotated outward.

C, the false and the true cords are blown widely open from the pressure of expired air. Note the "bowing" of the true cords.

D, after the initial release of pressure the true cords reapproximate, touching at one point, with partial closure of the false cords.

E and *F*, reopening. Note the irregular wavy outline of the true cords, the variations in width throughout their length and their generally flaccid appearance.

G, the second cycle of many repeated wavelike openings and closures begins.

has been to trap air within the tracheobronchial tree below the level of the larynx, as occurs primarily in cough but sometimes in thoracic fixation as well, the sudden release of this air under pressure produces a purposeless waving movement of the true cords (fig. 9). This simulates a rapid snakelike undulating movement in the anterior-posterior diameter and a to and fro movement from side to side taking place simultaneously. The cords obviously are in a state of relaxation, these movements apparently following no specific pattern and at times taking place with the cords more or less widely separated. The speed and excursion of the movements vary with the degree of air pressure that has accumulated during the maintenance phase of the closing cycle. Thus, in cough, when the accumulated pressure has been great and its release sudden, the cordal movements take on wide and rapid excursions of a rather wild type. On the other hand, in certain instances of thoracic fixation, especially when this takes place with the thorax contracted, so that the volume of air within it is considerably reduced and expiratory pressure therefore at a minimum, the movements of the cords during relaxation of the sphincter are slower, reduced in their period of excursion and continued for a shorter time.

If these movements are studied by sufficiently slowed motion pictures, single frames demonstrate that during the period of release the true cords have a wavy outline and vary in width from one point to the other throughout their length.

NERVE SUPPLY OF THE SPHINCTER MECHANISM

The motor nerve supply of the larynx is generally considered to be through the recurrent laryngeal nerves, with the exception of the cricothyroid and portions of the interarytenoid muscles. The former are supplied not by the recurrent but by the superior laryngeal nerves, and the latter, perhaps by both. Thus, the greater part of the sphincter mechanism of the larynx is under the control of the recurrent laryngeal nerves, but a portion of it at least is controlled by the superior laryngeal nerves. At any rate, it can be said with great assurance that in the presence of complete paralysis of a recurrent laryngeal nerve, when absolutely no movement of the affected true cord or its corresponding arytenoid is visible on phonation, an appreciable movement may take place within the structure of the false cord and the adjacent area during sphincteric activities. This is not sufficient to bring about complete closure but is appreciable, nevertheless, and certainly adequate to prove beyond all shadow of a doubt that the sphincteric mechanism is not under the complete control of the recurrent laryngeal nerves. For the reasons just given, this mechanism must be supplied in some small measure by the superior laryngeal nerve in addition to the recurrent

laryngeal. The muscle fibers supplied by the superior laryngeal nerves are probably those of the interarytenoid muscles, which blend into the false cords and extend along the lateral wall into the aryepiglottic fold.

FUNCTION OF THE SPHINCTER MECHANISM

Up to this point I have described the evolutionary beginnings of the larynx as a sphincter band, the anatomy of the organ in man and its method of closure. I have, however, only in a general way hinted at the physiologic function of this sphincter girdle. Its purpose is to isolate mechanically the respiratory organs below the larynx from the respiratory passage above. In so doing, the sphincter serves under varying circumstances to accomplish three main end results.

1. During swallowing it protects the lower passages from invasion by foodstuffs from above.

2. It makes thoracic fixation possible by trapping air within the thorax and at the same time preventing the entrance of additional quantities from above. This has far reaching effects in the use of the pectoral girdle group of muscles and also in the establishment of increased intra-abdominal pressure.

3. In cough it permits the momentary accumulation of increased intratracheobronchial pressure prior to the expulsive effort.

Each of these functions will be described in some detail.

Protective Action in Swallowing.—Changes in the anatomic structure of the primitive sphincter muscle band have taken place according to environmental needs. There has nevertheless been retained in almost every species some form of this primitive sphincter, which acts as a barrier to the passage of foodstuffs and other foreign material into the lower respiratory tracts, especially during swallowing. The muscular mechanism by means of which this sphincter-like closure takes place has already been described in a previous section on physiologic anatomy and need not be repeated. Certain laryngeal movements, however, are peculiar to swallowing and need not occur under other circumstances when the sphincteric action takes place. The movements peculiar to or at least most pronounced during swallowing require some consideration.

Through the action of the thyrohyoid, the palatopharyngeus and the stylopharyngeus muscles and the inferior constrictors of the pharynx, the entire larynx is elevated during deglutition, as does not necessarily occur during sphincteric closure for other purposes, to a position near the base of the tongue. This elevation, together with the forward movement of the cricoid and arytenoid cartilages and the puckering sphincteric closure of the aryepiglottic folds, has the effect of producing a large cavity posterior to the larynx for the reception of the bolus of food.

There is, therefore, a close anatomic association and synchronization between closure of the sphincter and the opening of the superior esophageal hiatus during swallowing. An added factor in the creation of this large funnel-like esophageal opening is the partial insertion of the esophagus into the cricoid cartilage and the cartilages of Santorini. As a result of this insertion, when the larynx is elevated for deglutition the anterior portion of the esophageal orifice is likewise elevated, thus aiding in the creation of the widely opened funnel-like aperture which receives the bolus of food. Direct observation of this region during swallowing reveals that the larynx, having been closed by approximation of the false cords as well as by the closed aryepiglottic folds, which are forced against the tubercle of the epiglottis, and the elevation of the entire larynx having been established, those structures previously recognized as the piriform sinuses have lost their identity as such and have become part of the single large esophageal hiatus. This requires some explanation. Examination of the larynx in the resting stage shows lateral to the aryepiglottic folds a depression on each side, the piriform sinuses. If probed each sinus is found to lead into the esophagus. Potentially these sinuses are one cavity, representing the lateral extremities of the upper orifice of the esophagus but apparently made into two isolated orifices by the projection backward of the mass of the cricoid cartilage against the posterior hypopharyngeal wall, which obliterates the midportion of the potential space. When the cricoid cartilage is displaced anteriorly, either by an esophagoscope or by swallowing, the potential space is recreated, so that it and the two piriform sinuses again fuse into one, creating an open mouth which is the upper extremity of the esophageal orifice. The sphincterically closed larynx lies anterior to this, and as food passes toward the superior esophageal orifice, its passage through the larynx is prevented by the tightly closed double layer of sphincter muscles.

Cessation of respiration takes place during swallowing and is involuntary, occurring as a result of reflexes arising in the pharynx from the presence of food. These stimuli pass to the central nervous system through the ninth nerve.

All I have said pertaining to protection of the lower respiratory passages by the sphincter during the swallowing of foodstuffs applies equally to prevention of inhalation of other foreign matter into the trachea. Those laryngeal reflexes which when stimulated by contact with foreign matter result in sphincteric closure are likewise stimulated when invasion is attempted by such material as saliva, nasal discharge, blood and mucus. This constitutes part of the role played by the larynx as "watchdog of the lungs," which has been mentioned so often by Chevalier and C. L. Jackson.[2] Infected material is thereby pre-

2. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937, p. 19.

vented from entering the lungs, so that the sphincter action of the larynx (in addition to the cough mechanism) plays a most important part in the prevention of pulmonary infection. In the absence of a functioning sphincter, the incidence of pulmonary sepsis, as would be expected, is greatly increased. Function is in abeyance during drug narcosis, in sleep and under other circumstances, as pointed out by the Jacksons in their numerous writings and even more recently by Applebaum.[3]

Control of Thoracic Fixation.—The second and equally important function of the sphincteric mechanism is its role in the production and maintenance of a fixed, immobile thoracic cage, which is essential under a variety of circumstances. For example, the muscles of the pectoral girdle arise, for the most part, from the ribs. In order that these muscles may effectively move the arms it is essential during certain arm movements that the ribs, representing the origin of these muscles, be fixed as firmly as possible. If this were not the case, attempts to depress the arms forcefully to the body against resistance, as a sailor does in hoisting sail, or to raise the body with the arms fixed, as in "chinning" on a bar, would result in the elevation of the thoracic cage instead of the lowering of the arms. Such elevation would bring the origin of the pectoral muscles on the rib cage closer to their point of insertion on the arm, thereby shortening the muscle and diminishing the effectiveness of its action. It is therefore important in activities of this sort that the rib cage be held down so as to lengthen these muscles as much as possible and to give them a fixed point of origin instead of a movable one from which they might contract. Such fixation could possibly be accomplished by the downward pull of the muscles of the abdominal wall and by the action of the intercostal muscles, but this would necessitate a greatly added expenditure of strength and energy. The substitute for this would be to fix the thorax in inspiration by a valve mechanism so that air can neither enter nor leave it, thereby preventing movements of the ribs. Such a mechanism is provided by the sphincter action of the larynx, and, naturally, since the larynx is completely closed, requires the temporary cessation of respiration. This fixation of the thorax is advantageous in that the rib cage not only lowers itself, establishing full physiologic length of the pectoral muscles, but, as has been indicated, it also insures the point of origin of the muscles becoming a steady one instead of one which constantly moves up and down.

Practically all animals which forcibly and independently use their forelimbs for climbing, squeezing, grasping and like movements have an

3. Applebaum, I. L.: The Respiratory Defense Mechanism, Am. J. M. Sc. **198**:356-366 (Sept.) 1939.

especially well developed laryngeal sphincter valve. From this point of view the larynx of man is very efficient, but in those mammals which swing from trees and require constant use of the forelimbs on which to suspend the body, the valvelike qualities may be even more highly developed.

The closure of the laryngeal sphincter, then, by preventing air from entering or leaving the thorax, establishes the position of the rib cage at a fixed point during use of muscles in and about the shoulder. With such a mechanism available in which egress and ingress of air can be controlled, it is at once apparent that the thorax can be fixed either with the lungs full of air and the ribs expanded or with the lungs relatively empty and the rib cage contracted and thus lowered, or at any intermediate stage between these two extremes. With the chest filled and the ribs expanded, the two ends of a pectoral muscle are brought closer together and the muscle shortened. This renders the muscle less efficient than when the thorax is emptied and the rib cage lowered. Under the latter circumstances, with the thorax emptied and the ribs contracted, the points of origin and insertion of the pectoral muscles are carried farther from each other, so that these muscles are lengthened and their strength proportionately increased. It is apparent, therefore, that muscular efforts of the forearm can be made more powerful by holding the breath, having first emptied the thorax and thereby lowered the ribs. Actually, such movements most often take place with the thorax fixed immobile in midposition. Whatever thoracic position is initially assumed maintains itself in status quo, because the closed laryngeal sphincter prevents the entrance of more air to expand the thorax further or the escape of air, which would allow it to contract.

Observations on the larynx under such circumstances reveal that the structure does not present similar reactions in every one but that certain variations can take place. Characteristically, in strong males, forceful movements of the forelimbs with the thorax partially emptied results in a very tight closure of both the true and the false vocal cords. If the effort is less forceful, complete action of the false cords does not take place, although partial closure may occur. In women who do not ordinarily carry out very powerful movements of the forelimbs and therefore do not require complete thoracic fixation, attempts to use the pectoral girdle forcefully may find the larynx in the ordinary position of respiration, or, with greater effort, partial or even complete closure of the true cords may take place. Sometimes even in frail women the false cords may partially or completely close with extremes of effort and the larynx present the same appearance as that in strong males.

The efficiency of this laryngeal mechanism in adding to the strength of the pectoral girdle can be easily recognized by observations on persons who lack such a mechanism, for example, in those breathing through tracheotomy tubes. Tracheotomized patients have no such laryngeal mechanism available and cannot effectively produce thoracic fixation. Their muscular efficiency is, as a result, profoundly diminished but can be considerably increased—at least momentarily—by corking the tube, provided the false cords above the tube can still be approximated.

The action of the laryngeal sphincter in fixation of the thorax has certain far reaching effects on circulation. It is common to observe during exaggerated physical efforts involving the arms or during thoracic fixation for any great muscular effort that the subject becomes cyanotic. Cyanosis may become extreme, with engorgement of the veins of the head and face. Cerebral symptoms, such as vertigo or even loss of consciousness, may ensue. This is especially marked if the thorax has first been filled with air and the rib cage contracted down against this positive intrathoracic pressure with no chance for its release because of the powerful sphincter action at the larynx. This increase in pressure within the thorax compresses the large veins and the auricle of the heart, as well as the capillary network, so that intrathoracic circulation is impeded. In addition, the sucking effort of negative intrathoracic pressure is lacking, for which reason the return flow of blood into the thorax does not effectively take place. This effect is very appreciable, and the pulse at the wrist may disappear. In persons with cardiovascular disease, such changes in circulation may lead to serious vascular accidents.

The thoracic fixation made possible by the sphincter action of the larynx is by no means limited to promoting efficiency in movements of the upper part of the arms. Its effects are vastly more far reaching than this. It plays a major role in a variety of other physiologic activities through its effect in making possible an increase in intra-abdominal pressure. The part played by thoracic fixation and the sphincter action of the larynx during the production of increased intra-abdominal pressure for a variety of purposes must therefore be described in some detail.

Production of Elevations of Intra-Abdominal Pressure.—Dependent at least in part on thoracic fixation is the ability to increase pressure within the abdomen. On a number of occasions indirect reference has been made to the role of the larynx in making possible elevations in intra-abdominal pressure. Such elevation of pressure within the abdomen is an essential part of the physiology of urination, defecation, childbirth, vomiting and other similar phenomena. It is brought about directly by contraction of the muscles of the abdominal wall but is aided in no small measure by the action of the thorax and diaphragm and controlled by the sphincter mechanism of the larynx. As an accom-

paniment of the contraction of the abdominal wall there takes place a deep inspiratory movement, which fills the lungs with air, expands the chest and immobilizes the ribs. This thoracic expansion is accompanied by a depression of the dome of the diaphragm, which, because of the fixation of the rib cage, tends to become fixed in its lowered position. Such fixation of the diaphragm in an extremely low position presents to the action of the contracting abdominal muscles a firm superior wall, sufficiently powerful not to be displaced by the increased pressure below it. None of this would be possible were it not for the sphincter action of the larynx, which prevents the escape of air from the lungs. The sphincter action in this instance consists of the usual approximation of the true and more especially of the false vocal cords. If these thoracic adjustments did not take place, then contraction of the abdominal wall would not so effectively increase pressure within the abdomen and its contained viscera. Instead, it would serve, at least partially, to elevate the diaphragm, force the air out of the lungs and collapse the thoracic cage. The pressure resulting from the contraction of the abdominal wall, therefore, would be dissipated into the chest and result in only a very slight, relatively ineffective increase in intra-abdominal pressure.

The effect on circulation of the fixation of the thorax under positive pressure as an accompaniment of increase in intra-abdominal pressure is not to be overlooked. It has been described previously, in the section on thoracic fixation.

The ability to elevate abdominal pressure is an important factor in vomiting, and in this the sphincter mechanism of the larynx is especially interesting. Vomiting is made easier if the stomach is distended with air, since the internal pressure of the stomach can under these circumstances be markedly increased by contraction of the abdominal wall. For this reason, the vomiting act is usually preceded by a deep inspiration, filling the lungs with air, expanding the thorax and lowering the diaphragm. Just before the expulsion of gastric contents the laryngeal sphincter is closed. This has been repeatedly observed laryngoscopically in patients recovering from anesthesia. With the sphincter of the larynx closed and with an accompanying simultaneous opening of the inlet of the esophagus, the stomach becomes filled with air by means of aerophagia. The laryngeal outlet remains shut, the thoracic cage squeezes down, increasing intrathoracic pressure, and the diaphragm, as the result of an inspiration, becomes depressed and fixed. Contraction of the abdominal muscles with the thorax and diaphragm fixed increases intra-abdominal pressure and presses the walls of the stomach together. This forces the gastric contents through the cardiac sphincter, which is relaxed, in contrast to the pyloric sphincter, which is closed. Toward the end of the expulsion of gastric contents or instantly after it, the sphincter of the larynx relaxes and air originally

inspired into the lungs is released under considerable pressure. This prevents the vomited material from entering the larynx.

Mechanism of Cough.—The sphincter action of the larynx is particularly well demonstrated in the cough reflex. A coughing cycle consists of several stages. During the first stage the larynx is widely opened, so that a deep sudden inspiratory effort can be made and the lungs filled with air. The cough becomes proportionately more forceful as the amount of inspired air increases. The next step is designed to increase intrathoracic pressure as much as possible and is accomplished by a valvelike closure of the larynx, in which both the true and the false cords tightly approximate. I have already discussed the relative lack of ability of the true cords to prevent the egress of air from the thorax, so that this function becomes essentially a matter of the efficiency of closure of the false cords. These effectively prevent the escape of intrathoracic air, which has accumulated rapidly and under great pressure. This is a vital part of the cough mechanism. Animals which do not possess functioning ventricular bands suffer a marked diminution in the power of their cough reflex.

In cough the laryngeal sphincter girdle comes into action and hermetically seals the larynx. Not only do the false cords approximate, but the arytenoids move forward to fill the posterior gap, and all those laryngeal movements already described in the discussion of the sphincteric closure of the false cords can be seen. The thorax compresses itself against the air trapped by the sphincter within the confines of the tracheobronchial tree, and this compression within a fixed space greatly increases the pressure of the retained air, forcing added quantities out of the lungs into the bronchi and the trachea, so that accumulated subglottic pressure becomes very great indeed. Suddenly the laryngeal sphincter flies open. Air, under the high pressure which has accumulated below the glottis, escapes in a more or less explosive fashion, and this is accentuated by further contraction of the thoracic cage. The sudden egress of this large quantity of air under great pressure is the cough and carries with it the foreign matter that is to be expelled.

In many instances this sudden forceful wide opening of the larynx is followed by a reaction in which the true cords may be seen to rebound momentarily almost to the midline and then to reassume their neutral respiratory position.

EFFECT OF LOSS OF THE SPHINCTER VALVE

It may well be asked why, if this sphincter mechanism plays so vital a role in human physiology, do patients who have had the sphincter girdle completely removed, as in total laryngectomy, survive so well? In the first place, complete laryngectomy so radically alters normal

anatomic situations that many factors requiring the action of the sphincter are no longer existent. For instance, in swallowing the lower airway is not exposed to the danger of invasion by foodstuffs, since its superior orifice is now outside of the neck, no longer in close proximity to the pharynx and the superior esophageal hiatus, and therefore not in the route of passage of material that is being swallowed. The tracheal orifice having thus been removed from harm's way, a protective sphincteric action during swallowing is, after laryngectomy, not necessary. For the same reason, the trachea can no longer be invaded by saliva and infected postnasal discharges, so that the sphincter loses its importance as the "watchdog of the lungs." Its absence under these circumstances is again compensated for by the new position of the tracheal inlet.

In the matters of thoracic fixation, power to increase intra-abdominal pressure and ability to carry out effective cough, there can be no question that the loss of the laryngeal sphincter after total laryngectomy is sorely felt. While the relative inefficiency of these various functions after the surgical loss of a sphincter proves inconvenient and has far reaching deleterious effects on bodily economy and physiology, the diminution in effectiveness of these functions is nevertheless not vital to the maintenance of life. For this reason the loss of the laryngeal sphincter is reasonably well tolerated. There is, nevertheless, loss of power in the use of upper extremities, and there may be difficulty and loss of efficiency, especially at first, in urination and defecation as well as in all other activities requiring thoracic fixation or increases of intra-abdominal pressure. Likewise, cough proves less effective, the difficulty in expelling sputum from the depths of the lower respiratory passages being especially noticeable.

The reasonable efficiency with which surgical loss of the sphincter is tolerated becomes possible only because the surgeon, recognizing the function of the organ, has instituted measures to compensate for its loss. The value and importance of the laryngeal sphincter cannot, therefore, be gaged entirely by the effect of its surgical removal.

SUMMARY

The larynx has developed not primarily as a sound-producing organ but rather as a sphincter valve to isolate the upper from the lower respiratory passages. It first appeared as a circular muscle band in the lungfish, to prevent sea water from invading the lungs. A cartilaginous supporting framework made its appearance in reptiles, to permit the maintenance of a patent lumen despite the prolonged presence in the adjacent esophagus of a huge bolus of food. This framework has been retained throughout the evolutionary cycle. The muscular

sphincter came to lie as a horizontal band within the lumen of the cartilaginous structure and ultimately in certain mammals, such as man, became separated by a fissure into lower and upper divisions, the true and the false vocal cords. The latter constitute the principal element of the sphincter girdle. They are composed for the most part of striped muscle tissue, chiefly the mass of the superior division of the thyroarytenoid muscle. Some of the fibers of the superior division pass laterally around the arytenoid to join the interarytenoid muscles, and, likewise, fibers of the interarytenoid muscles pass anterolaterally into the false cord. This arrangement forms the "arytenoid sling," which constitutes an important muscular element in the formation of the sphincter girdle. Its closure is accompanied by a "foreshortening" of the length of the interior of the larynx by a shifting forward of the cricoid cartilage and the arytenoids and a shifting backward of the thyroid. The details of the muscular action that makes this closure possible are described. The true vocal cords are composed essentially of the inferior division of the thyroarytenoid muscles, and these play a minor role in the sphincter action.

Closure of the sphincter mechanism consists of a tight approximation, first of the true cords then of the false cords, the posterior dehiscence being filled in by the mass of the arytenoids, which tilt forward. Release of the sphincter is accompanied by a purposeless waving motion especially of the true cords.

A second sphincter, composed laterally of aryepiglottic folds, posteriorly by the mass of the arytenoids and anterosuperiorly by the epiglottis, is described.

It is pointed out that the sphincter mechanism is controlled not only by the recurrent laryngeal but in some small degree by the superior laryngeal nerve as well.

The purposes of the sphincter mechanism are:

1. To close off the entrance into the lower respiratory tract during swallowing and thereby prevent invasion of this tract by foodstuffs from above.
2. To permit thoracic fixation by trapping air within the thorax and by preventing the entrance of additional quantities from above. This has far reaching effects in the use of the pectoral girdle and in the establishment of increased intra-abdominal pressure.
3. To permit the momentary accumulation of increased intratracheobronchial pressure before the expulsive effort in cough.

The effect on these functions of the loss of the sphincter apparatus, as after laryngectomy, is briefly discussed.

PHOTOGRAPHIC STUDY OF THE LARYNX BY MIRROR LARYNGOSCOPY

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While marked advances have been made in photographing those regions of the body that are readily accessible to the camera and this method of making records is generally employed, photography of the larynx has not been developed so extensively until recently. This has not been due entirely to a lack of interest in the subject.

In 1860 Czermak made some studies of photography of the larynx. Solis-Cohen, Elsberg and others became interested in the subject, but little was accomplished until 1883, when Lennox Browne [1] presented a paper on this subject, together with some excellent photographs. He appeared to lack enthusiasm, however, for he stated:

> I do not anticipate that photography of the larynx can be extended beyond the boundary of physiology. To expect photographs from life of pathologic conditions is plainly unreasonable, since those we have seen could only have been obtained by elaborate and costly machinery, and, above all, from a subject possessed of unusual, indeed, in my experience, of unequaled knowledge of what was our goal, and skill and endurance necessary for its attainment.

One year later, in 1884, Thomas R. French,[2] of Brooklyn, read before the Section of Laryngology of the International Medical Congress, held at Copenhagen, Denmark, a paper on laryngeal photography and presented two portfolios of remarkable photographs of normal and diseased larynxes. In describing his method of photography, he stated: "Our object has been not only to obtain good photographs but to devise an easy method by which any expert laryngoscopist can photograph the larynges of his patients in everyday practice."

The subject was practically forgotten when Garel,[3] of Lyons, France, devised an apparatus to take stereoscopic pictures. These represented an outstanding contribution to the field of laryngeal photography. In

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Presented in part on a motion picture program at the Forty-Fifth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology, Cleveland, Oct. 10, 1940.

1. Browne, L.: Photography of the Larynx and Soft Palate, Brit. M. J. **2**: 811, 1883.

2. French, T. R.: On a Perfected Method of Photographing the Larynx, New York M. J. **40**:653, 1884.

3. Garel, J.: Nouvel appareil perfectionné pour la photographie stéréoscopique du larynx sur le vivant, Rev. de laryng. **40**:249, 1919.

1923 I began to use the Garel camera. The results were presented before the American Laryngological, Rhinological and Otological Society in 1924.[4] While this method gave the best results obtained up to that time, certain difficulties with illumination appeared insurmountable with the materials at hand, and the use of the method was largely discontinued.

The introduction of the small motion picture camera gave a new impetus to laryngeal photography. The major difficulties had been the relative inaccessibility of the larynx and inadequate illumination. Progress has been dependent on the securing of a better exposure of the larynx, on improved photographic equipment and on the development of faster films and more compact and more powerful electric light bulbs. While the black and white film gave satisfactory results in studying laryngeal motility, the introduction of the color film aided immeasurably in depicting pathologic changes.

In 1930 Russell and Tuttle[5] devised an apparatus, the fonofaryngoskop, and published the results they obtained with color movies of vocal cord actions.

Heatly[6] in 1931 reported on motion picture studies of the larynx which were secured by direct laryngoscopic procedure. A quartz rod was used to aid in illuminating the larynx. While the results were interesting and were the first of studies by this method ever recorded, there was inadequate illumination to secure clear definition and sufficient focal depth.

In 1933 LeJeune[7] presented cinematographic studies made with a suspension method, and in subsequent presentations[8] he exhibited the results of further observations and the use of color films in depicting laryngeal lesions. His contributions have been outstanding and have greatly stimulated interest in this subject, not only in the use of suspension laryngoscopy but also in direct and indirect laryngoscopic methods.

In 1937 Pressman and Hinman[9] presented a simple technic for photographing the larynx by direct laryngoscopic procedure. They equipped the laryngeal speculum with carriers for twin lights, which

4. Clerf, L. H.: Photography of the Larynx, Ann. Otol., Rhin. & Laryng. **34**:101, 1925.

5. Russell, G. O., and Tuttle, C. H.: Color Movies of Vocal Cord Actions an Aid to Diagnosis, Laryngoscope **40**:549, 1930.

6. Heatly, C. A.: Motion Picture Studies of the Larynx, Ann. Otol., Rhin. & Laryng. **40**:434, 1931.

7. LeJeune, F. E.: Suspension Cinematography of the Larynx, Arch. Otolaryng. **18**:70 (July) 1933.

8. LeJeune, F. E.: Motion Picture Study of Laryngeal Lesions, Surg., Gynec. & Obst. **62**:492, 1936.

9. Pressman, J. J., and Hinman, A.: A Simple Technic for Taking Motion Pictures of the Larynx in Action, Arch. Otolaryng. **26**:526 (Nov.) 1937.

provided adequate illumination and made possible excellent results. This principle of illumination has been modified by Holinger and now is generally accepted for photographing the larynx by direct laryngoscopic means.

Tucker [10] and Lell [11] photographed the larynx by mirror laryngoscopy, using an independent source of illumination. The light rays were reflected into the patient's mouth with a head mirror controlled by the laryngologist, who also held the laryngeal mirror in position. The camera was operated by an assistant and was independent of the source of illumination, the head mirror and the laryngeal mirror.

In an endeavor to secure motion pictures of the larynx without obscuring any part of the structure with a spatula or hampering its

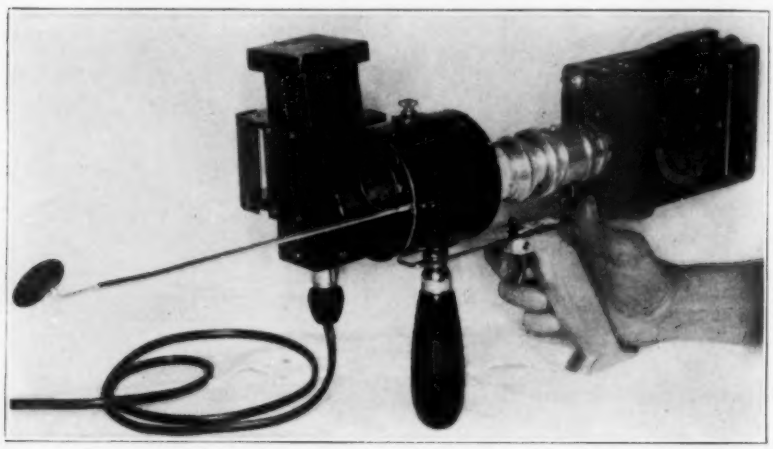

Fig. 1.—Photograph of unit used in laryngeal photography. The entire equipment is supported by the operator's right hand; the shutter release is operated by the index finger.

movements by suspension or fixation methods and at the same time to provide a practical yet simple apparatus, which could be readily employed with a minimum amount of equipment and personnel and which would require only a short time to operate and would give uniformly good results, I devised a photographic unit (fig. 1) which embodied the principle of the Garel camera. A field for improvement which was suggested was the development of a means for increasing focal depth. All of the photographs taken by mirror laryngoscopy up to this time exhibited a very narrow plane of the larynx in focus. If the vocal cords were in

10. Tucker, G.: A Technique for Motion Picture Photography of the Larynx, Tr. Am. Laryng. A. **61**:259, 1939.

11. Lell, W. A.: Motion Pictures of the Human Larynx, Arch. Otolaryng. **30**:344 (Sept.) 1939.

focus, the structures proximal and distal to them were indistinct. This could be corrected only by delivering more light to the larynx, so that the size of the diaphragm opening could be decreased.

DESCRIPTION OF APPARATUS

In the accompanying drawing (fig. 2) *A* and *B* represent the camera and the lens, while *C* contains the source of illumination and *D*, the condensing lenses and reflecting surface. With the laryngeal mirror attached, the entire equipment constitutes an inflexible unit.

Any magazine camera may be used. I have employed the Eastman magazine camera and the Bell-Howell Filmo 141-B equipment. A Kodak

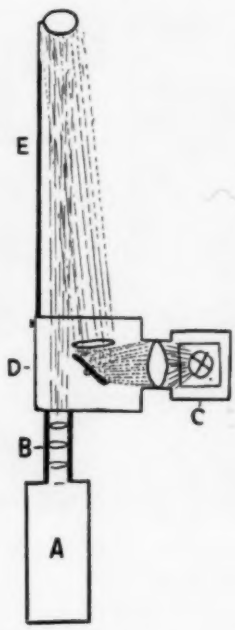

Fig. 2.—Schematic drawing of camera with source of illumination and laryngeal mirror attached for laryngeal photography. *A* represents the camera; *B*, the lens; *C*, the lamp house; *D*, the chamber supporting the mirrors and condensing lens, and *E*, the laryngeal mirror.

Anastigmat 2½ inch (6.3 cm.), F. 2.7 lens is used. It is probable that a 3 inch (7.6 cm.), F. 4.5 lens would give better results. In the average case the diaphragm opening used is F. 8.

The source of illumination is a small 200 watt projection lamp, enclosed in an insulated lamp house. The lamp is operated by a foot switch. The light rays are condensed by a convex lens and reflected by a round mirror, 1½ inches (3.8 cm.) in diameter, through a second convex lens to the laryngeal mirror. The handle of the mirror (*E*) is secured in a small slot in the chamber (*D*) with a thumb screw.

A no. 6 laryngeal mirror with a diameter of 1 inch (2.5 cm.) is commonly employed for adults. Larger mirrors may be used. For children, size 5 or 4 should be utilized. The distance from the center of the laryngeal mirror to the photographic lens is 11 inches (28 cm.). Further experience may indicate that this distance should be decreased.

The entire apparatus is supported by a pistol-like grip, held in the right hand, with the right index finger controlling the shutter trigger. The patient's tongue is held by the operator's left hand, the camera is held and operated by the right hand and the light is controlled by a foot switch. In certain cases it may be necessary to have an assistant depress the tongue with a depressor.

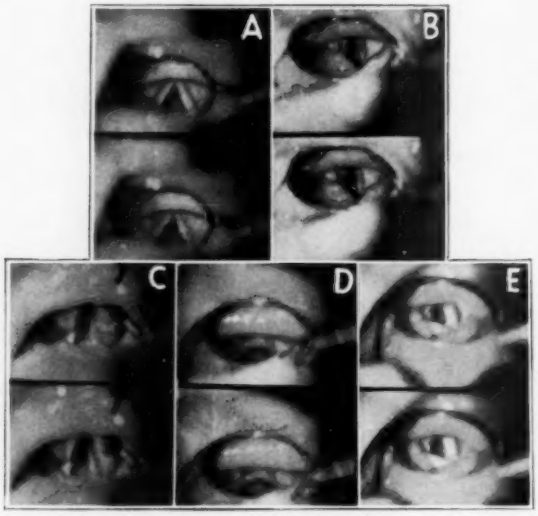

Fig. 3.—Illustrations of laryngeal images, taken by mirror laryngoscopy, reproduced from a 16 mm. Kodachrome film:

A, inflammatory thickening of the left vocal cord; *B*, paralysis of the right vocal cord; *C*, extensive carcinoma involving the entire right vocal cord; *D*, web in the anterior part of the larynx after successful removal seven years previously of a carcinoma from the anterior commissure by laryngofissure, and *E*, cicatricial changes in the larynx in a case of arrested tuberculosis.

METHOD OF OPERATION

Since the mirror cannot be moved independently of the camera or of the source of illumination, it is necessary to adjust the angle of the mirror so that a satisfactory view of the larynx may be secured along the axis of the mirror handle. The mirror then is attached to the camera and its position checked with the view finder. The approximate distance between the laryngeal mirror and some part of the patient's larynx, preferably the vocal cords, is determined, and the lens is then focused

for this additional distance. Fogging of the mirror is prevented by dipping the mirror in hot water immediately before using. The patient is seated in the ordinary examining chair, and no support for the head is used. The pharynx is sprayed with a 1 per cent solution of pontocaine hydrochloride. Since the photographic lens must be so placed as to secure the best view of the reflected image in the laryngeal mirror, it is necessary for the operator to view the mirror from the left side of the camera. After several trials, this slight angle can be readily compensated for.

Ordinary Kodachrome type A film is used at a normal speed of 16 frames per second. The lens stop usually employed is F. 8. This gives a focal depth of about 1½ inches (3.8 cm.) and brings a great part of the larynx in focus. In the accompanying black and white photographic prints (fig. 3), enlarged from a 16 mm. color film, the lesions are in the same plane as the vocal cords. It will be observed that structures proximal to this plane also exhibit fair definition.

SUMMARY

A simple apparatus for photographing the larynx by mirror laryngoscopy is presented. It may be employed readily, requiring no additional personnel to operate, and gives uniformly good results in a majority of patients whose larynxes can be visualized by mirror laryngoscopy.

Mr. J. W. Robbins, of Philadelphia, gave technical assistance in the development of the laryngeal camera and is now carrying out additional improvements.

RIEDEL'S STRUMA

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Of increasing interest to both the pathologist and the surgeon is the relatively infrequent involvement of the thyroid gland commonly known as Riedel's struma. The condition is so named because Riedel was the first to describe it, in 1896.[1] Hashimoto in 1912 described an involvement of the thyroid gland characterized by marked lymphocytic infiltration, to which he applied the term "struma lymphomatosa."[2] After a study of cases under his observation Ewing[3] came to the conclusion that Riedel's struma and struma lymphomatosa are different stages of the same disease, whereas other writers, especially Graham and his associates, maintained that they are two separate disease entities and in no way related.[4] Graham expressed the belief that Riedel's struma more nearly approaches true inflammation than does the lesion described by Hashimoto. Some authors have declared it to be a nonspecific granulomatous inflammation.[5] Struma lymphomatosa, on the other hand, is considered to be a degenerative change in the thyroid gland without evidence of an acute inflammatory process.[6] Various points of differentiation, clinical and pathologic, are stressed by certain authors.[7]

From the Department of Otology, Rhinology and Laryngology of the University of Illinois College of Medicine.

1. Riedel: Verhandl. d. deutsch. Gesellsch. f. Chir. **25**:101, 1896.

2. Hashimoto, H.: Arch. f. klin. Chir. **97**:219, 1912.

3. Ewing, J.: Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1934, p. 961.

4. (*a*) Graham, A.: West. J. Surg. **39**:681 (Sept.) 1931. (*b*) Graham, A., and McCullagh, E. P.: Atrophy and Fibrosis Associated with Lymphoid Tissue in Thyroid: Struma Lymphomatosa (Hashimoto), Arch. Surg. **22**:548 (April) 1931. (*c*) Polowe, D.: Struma Lymphomatosa (Hashimoto) Associated with Hyperthyroidism, ibid. **29**:768 (Nov.) 1934.

5. Schultz, O. T.: Tr. Chicago Path. Soc. **12**:313 (June) 1927.

6. Clute, H. M.; Eckerson, E. B., and Warren, S.: Clinical Aspects of Struma Lymphomatosa (Hashimoto), Arch. Surg. **31**:419 (Sept.) 1935. Graham and McCullagh.[4b]

7. (*a*) Gilchrist, R. K.: Chronic Thyroiditis, Arch. Surg. **31**:429 (Sept.) 1935. (*b*) McClintock, J. C., and Wright, A.: Ann. Surg. **106**:11 (July) 1937. Graham.[4a]

While it is not our purpose to take sides in this controversy, nevertheless, we feel, as Graham has said, that if case reports are to be of any value, an attempt should be made to distinguish between the two conditions.[8]

Riedel's struma usually occurs in persons around the age of 40. The distribution between the sexes is about equal. The outstanding condition observed is a diffuse, smooth and hard enlargement of the thyroid gland, usually involving one lobe to a more marked degree than the other. The extreme hardness of the tumor, described by Riedel as iron hardness, is characteristic. Tenderness to palpation may be complained of. Since in the majority of instances the inflammation spreads to the adjacent tissues, fixation of the gland results in over one half of the cases. The skin remains free. The regional lymph nodes remain uninvolved. As a result of connective tissue infiltration about the larynx, trachea and adjacent blood vessels and nerves, symptoms resulting from impaired function of these structures occur. Dyspnea, dysphagia or a feeling of pressure in the neck are some of the earliest symptoms which cause the patient to seek medical aid.[9] Hoarseness due to involvement of the inferior laryngeal nerve is not an uncommon manifestation. Occasionally, as in the case reported, the patient may complain of drawing pains along the side of the neck, more severe on turning the head. Objective signs of long-standing goiter, either as a diffuse adenoma or as adenomatous nodules, are frequent. The average duration of symptoms is about seven months.

A diagnosis of Riedel's struma is a difficult one to make. It should be considered only after other possible involvements have been ruled out. The symptoms and physical findings already discussed vary with the extent of involvement of the gland and the degree of extension to surrounding structures. The basal metabolic rate is not high as a rule. The white blood cell count averages 10,000 to 15,000.[7a] Mild toxic symptoms may be present. Roentgenograms may show areas of calcification in the gland, a condition which may be present in other thyroid diseases. Their chief value lies in showing the degree of tracheal compression or the destruction of tracheal rings.

In the differential diagnosis first and foremost to exclude is neoplasm. One author's contention that a definite diagnosis of this disease can be made only microscopically is a justifiable one.[10] Even then the decision may be doubtful, as demonstrated by a case mentioned by Clute and Lahey, in which the pathologist made a diagnosis of

8. Graham, A.: Personal communication to the authors.
9. Bruce, H.: Ann. Surg. **95**:562 (Oct.) 1931.
10. Wingate, H. F.: Brit. J. Surg. **17**:264 (Oct.) 1929.

Riedel's struma and the condition later turned out to be malignant.[11] In carcinoma of the thyroid the gland is usually nodular, whereas in this disease the gland is often smooth and the borders indefinite. Dysphagia is more suggestive of malignant growth than of chronic thyroiditis.[12] Syphilis, tuberculosis, actinomycosis and woody phlegmon of the thyroid likewise enter into the differential diagnosis.[13]

On gross examination the thyroid gland is enlarged, hard in consistency and of a faint pink or gray color on cut section. The gland is adherent to surrounding structures, but the skin is free. On cutting, the surgeon can feel increased resistance to the knife blade. In contrast to the microscopic picture presented in struma lymphomatosa, fibrosis is a prominent feature of Riedel's struma. The colloid acini present a varied aspect. They may be of the fetal type or of the hypertrophic type, or they may be in the final stages of regression indicative of adenomatous changes in the gland. Fibroblastic proliferation with areas of hyaline degeneration is abundant. The remaining thyroid cells may be choked off into isolated islands by the fibrous proliferation. Pseudogiant cells may be formed. It is an active fibrosing process with enlargement of the gland and extension to and involvement of neighboring structures.[14]

Opinions as to the best method of treatment vary. Since focal sources of infection, in some instances, seemed to play a role in causation of the disease, a careful search for such foci should be made and these eliminated. Particular attention should be paid to the teeth, sinuses and tonsils. Regression of the disease when only small portions of the gland were removed have been reported.[15] Benson recommended surgical removal with as complete a thyroidectomy as possible.[16] On the other hand, Portman and Crile expressed the opinion that when a clinical diagnosis can be made, roentgen therapy is the treatment of choice.[17] Renton and his associates cited 5 cases in which progress of the disease was arrested by the use of radium.[18] Deep roentgen therapy combined with partial removal of the gland has proved beneficial in the case we have reported here. When pressure symp-

11. Clute, H. M., and Lahey, F. H.: Ann. Surg. **95**:493 (April) 1932.

12. Tearman, C. H.: Illinois M. J. **66**:475 (Nov.) 1934.

13. Eisen, D.: Am. J. M. Sc. **192**:673 (Nov.) 1936.

14. Means, J. H.: The Thyroid and Its Diseases, Philadelphia, J. B. Lippincott Company, 1937, p. 500.

15. Bernard, A. F., and Smith, R. P.: Brit. J. Surg. **13**:93 (July) 1925.

16. Benson, B. B.: Am. J. Surg. **27**:361 (Feb.) 1935.

17. Portman, U. V., and Crile, G.: Diagnosis and Treatment of Diseases of the Thyroid Gland, Philadelphia, W. B. Saunders Company, 1932, p. 345.

18. Renton, J. M.; Charteris, A. A., and Heggie, J. F.: Brit. J. Surg. **26**:54 (July) 1938.

toms become manifest, surgical intervention may be necessary. The operation is rendered difficult because of extension of the fibrosing process to surrounding neck structures, resulting in extensive adhesions, loss of anatomic landmarks, excessive hemorrhage and respiratory embarrassment.

We deem the case we have observed worthy of reporting because of the comparative rarity of the disease and because of the characteristic features attributed to Riedel's struma which are demonstrated.

REPORT OF A CASE

History.—On July 7, 1939, a white man, aged 57, complaining of a painful swelling on the right side of his neck, was admitted to the hospital. He stated that as long as he could remember he had had an enlargement on the right side of his neck which had caused him no trouble until six months before admission, when his wife noted a gradual increase in its size, which had been progressive since. Two weeks before he was admitted to the hospital he began having pains, which radiated up the right side of his head and neck and which became more pronounced when he turned to the right. In the past, he claimed, his general health had been good. He had had frequent general physical examinations, at which times his physicians had declared him to be in good health. His history of systems was irrelevant. His family history revealed nothing of diagnostic significance except diabetes mellitus in the mother.

Physical Examination.—The patient was a well nourished white man of 57. The upper teeth were replaced by a denture, and the lower teeth were in good condition. The tonsils were not unusual. Reflexes were normal in response. The right lobe of the thyroid was large, very firm to palpation and intimately connected with the surrounding structures. The skin was freely movable. No nodes were palpable. The left lobe was slightly enlarged. The heart, lungs, and abdomen were normal.

The temperature was 98.6 F. The blood pressure was 146 systolic and 90 diastolic. The pulse rate was 85. The urine was normal. Blood studies showed hemoglobin content of 80 per cent, a red blood count of 4,300,000 and a white blood count of 15,800. The differential count showed 77 per cent neutrophils, 17 per cent small lymphocytes and 4 per cent large lymphocytes. The basal metabolic rate was —3.8 per cent. The Wassermann reaction was negative. Roentgenologic examination of the chest gave negative results. Roentgenologic examination of the neck showed areas of calcification in the thyroid gland (fig. 1). The trachea was slightly deviated to the left, but no compression was evident.

The tentative diagnosis was a malignant growth of the thyroid gland. A biopsy was performed on July 7, 1939. On microscopic examination the section showed thyroid tissue with acini in various stages of regression. Fibrosis was marked, resulting in obliteration of most of the acini. Aggregations of cells resembling giant cells were present. The clinical picture resembled that of chronic thyroiditis (Riedel's struma).

Treatment.—Irradiation of the tumor mass was decided on. However, after the third treatment the patient began complaining of increasing discomfort, and the mass on the right showed an increase in size and was tender to palpation. This form of treatment was therefore discontinued and an operation decided on.

Fig. 1.—Lateral view of the neck, showing areas of calcification.

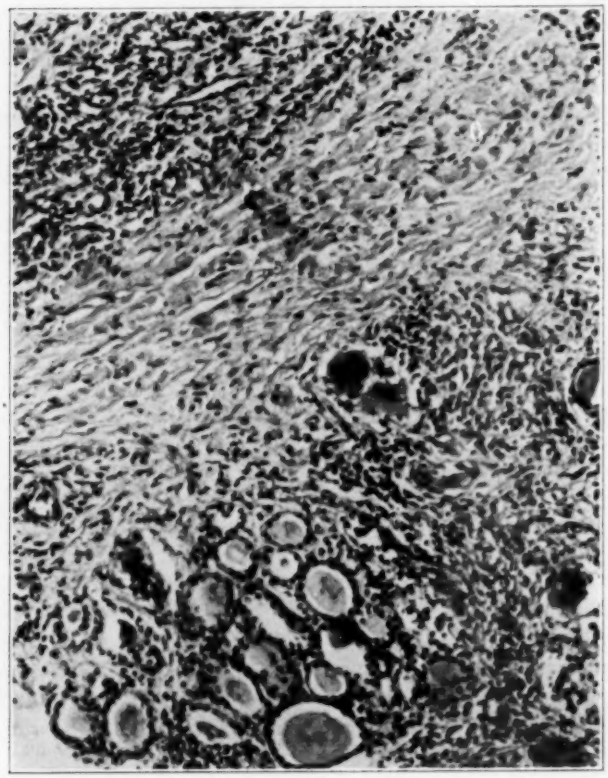

Fig. 2.—Photomicrograph of an area of marked fibrosis.

Operation.—On July 13, 1939, the patient was operated on. The operation was performed under ethylene analgesia and procaine hydrochloride infiltration. A linear incision on the right side of the neck, anterior to the sternocleidomastoid muscle, was made. A large indurated mass, intimately connected with the adjacent structures, increased the difficulty of the operative procedure. As much of the right lobe was removed as was possible.

Pathologic Report.—The specimen measured 9 by 6 by 2 cm. On section the color was yellowish gray, indicative of dense fibrous tissue. Nodules of light

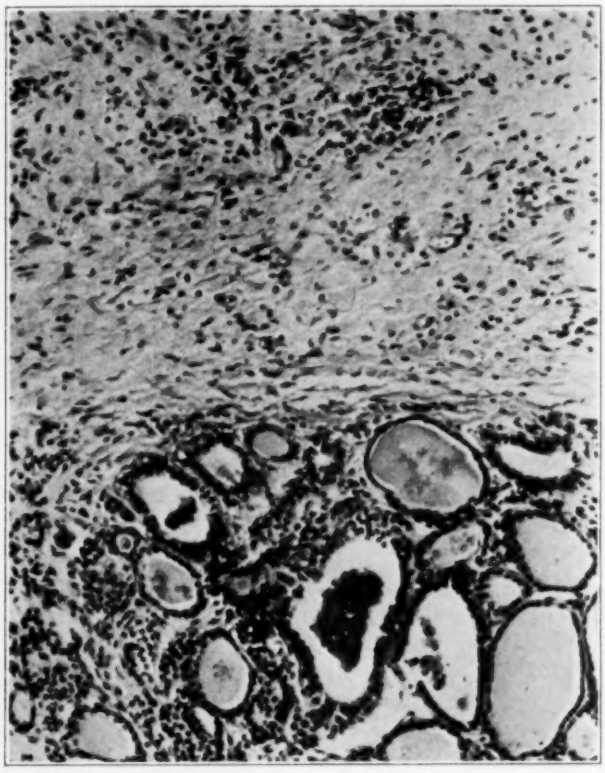

Fig. 3.—Photomicrograph of an area showing degeneration of acini and compression of acini by fibroblastic proliferation.

tan thyroid tissue, soft to the touch, could be noted. Portions of these were calcified. Located at one pole of the mass was an entirely calcified area measuring 2 by 1 by 1 cm.

Microscopic examination of one section revealed numerous areas of degenerating thyroid tissue showing acini compressed by marked fibroblastic proliferation. Another block revealed an area of normal-appearing thyroid tissue with a border of dense hyalinized connective tissue. The latter contained vesicular spaces suggesting deposits of cholesterol. In some areas accumulations of cells resembling giant cells could be seen. These were few. Figures 2 and 3 show representative areas of the sections described.

Pathologic Diagnosis.—Chronic diffuse thyroiditis (Riedel's struma).

Further Course.—The patient made an uneventful recovery and was discharged on July 18, 1939. He returned to us one month later, complaining of a slight increase in size of the mass on the left side of his neck. Examination revealed a well healed scar on the right side of the neck, with an area of induration still present. The left lobe of the thyroid showed a moderate increase in size over that noted at the first examination. Since no reaction had occurred on the left side during the first course of irradiation, this form of treatment was again resorted to. It was deemed advisable also to irradiate the site of the operation in order to reduce further any activity there. Both thyroid areas were approached through portals 13 cm. by 17 cm., covering the area from the inferior border of the mandible to the level of the clavicle, and from the midline outward. The total dose to each portal was 636.5 roentgens, with a combined total dose of 1273 roentgens (approximately 2.8 times the erythema dose).

A gradual reduction in size of the tumor mass on the left was noted. Clinically the patient showed improvement. He has been followed at regular intervals and up to the time of the last examination, in May 1940, has had no recurrence of symptoms.

SUMMARY

The need of differentiating between struma lymphomatosa and Riedel's struma is discussed. The clinical and pathologic findings ascribed to Riedel's struma are enumerated. Methods of treatment varied with different authors. A case of Riedel's struma is described and the method of treatment given. Good results were obtained by resorting to surgical procedure combined with radiation therapy.

CHORDOMA OF THE BASIOCCIPUT AND BASISPHENOID

REPORT OF FOUR CASES

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MONTREAL, CANADA

Before 1935, cranial chordoma was considered a rare tumor. In that year Mabrey (1935)[1] found that only 47 instances had been reported since Müller (1858)[2] first correctly identified the neoplasm as a growth from the notochordal remnants. In the years since 1935, however, this figure has been raised to approximately 85 (including cases collected by Peers,[3] 1938, and single cases reported since 1938).

We wish to add the 4 cases studied at the Montreal Neurological Institute and the Montreal General Hospital, because they represent the two major subdivisions of chordoma of the basiocciput and basisphenoid, viz., that in which the tumor presents in the nasopharynx —one fifth of the cases—and that in which it is confined to the cranial cavity and its walls. In addition, we wish to point out the value of stained smear preparations in the diagnosis of this tumor, especially when it presents in the nasopharynx. We shall also comment on the treatment of these growths.

In most cases chordoma of the basiocciput and basisphenoid grows predominantly within the cranial cavity. We have seen 2 such cases.

REPORT OF CASES

CASE 1.—P. M., a man aged 26, was admitted to the Montreal General Hospital to the service of Dr. W. Penfield.

For two and a half years this man's vision and hearing had diminished, so that he was almost completely blind and deaf. Also the right fifth, sixth, seventh, ninth,

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From the Department of Neurology and Neurosurgery of McGill University and the Montreal Neurological Institute.

1. Mabrey, R. E.: Chordoma: A Study of One Hundred and Fifty Cases, Am. J. Cancer **25**:501, 1935.

2. Müller, H.: Ueber das Vorkommen von Resten der Chorda dorsalis bei Menschen nach der Geburt und über ihr Verhältniss zu den Gallertgeschwülsten am Clivus, Ztschr. f. rat. Med. **2**:202, 1858.

3. Peers, J. H.: Spheno-Occipital Chordoma, Am. J. Cancer **32**:221, 1938.

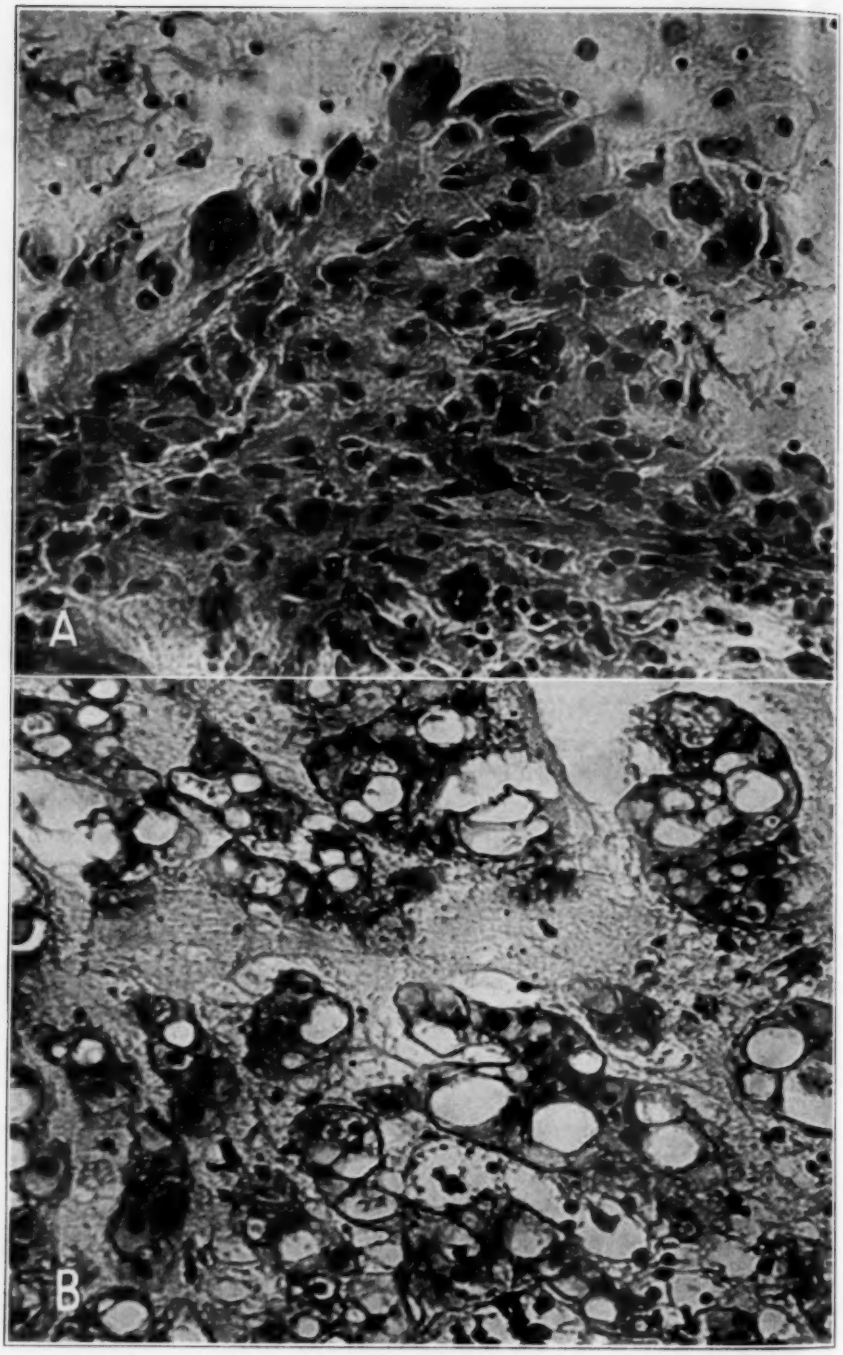

Figure 1

(See legend on opposite page)

tenth and twelfth cranial nerves were partially involved. There were hyperreflexia on the right side and partial loss of the sense of position.

There was no roentgen evidence of cerebral ventricular shift, but the clinoid processes were eroded and the floor of the sella turcica was rough.

At operation a reddish irregular encapsulated tumor filled the cerebellopontile angle, pushing upward against the tentorium and stretching the right glossopharyngeal roots below. Posteriorly it pushed into cerebellar substance. Pieces of friable tumor material were curetted away, but complete removal of the growth was not attempted. The patient died on the second day after the operation.

At autopsy a large nodular whitish pink mass of opaque cheesy tumor was found covering the basiocciput and basisphenoid, replacing the dorsum sellae and resembling in appearance that which had been removed at operation. It was more extensive on the right side. The optic nerves were thinned out over its cephalic end; caudalward it stretched the vagus and the glossopharyngeal nerve. It deformed adjacent parts of the brain. The pituitary body lay anterior to it and was unaffected.

In sections routinely prepared for biopsy and autopsy, the tumor is seen to be composed of large cells, occasionally multinucleated, with many nuclear inclusion bodies. No mitotic figures are seen. The abundant granular cytoplasm contains many vacuoles. Smaller cells, which are thought to be more embryonic forms, are present. There is a noteworthy absence of connective tissue, of intercellular matrix and of blood vessels.

The diagnosis was chordoma.

Case 2.—A. D., a man aged 21, was admitted to the Montreal Neurological Institute to the service of Dr. A. R. Elvidge.

For three years this patient had had deep occipital pain on the left side and dizziness when he shook his head. For six months, the shaking of the head had also produced a heavy feeling in the upper part of the left jaw.

The only definite physical abnormalities were slight edema of the left optic disk and a fine lateral nystagmus, more marked on gaze to the left.

Pneumographic studies showed posterior displacement of the fourth ventricle, with obliteration of the interpeduncular and pontile cisterns. The films showed no change in the basiocciput or the basisphenoid.

At operation gray tapioca-like neoplastic nodules were found between the right fifth and seventh cranial nerves. As much as possible of the neoplastic material was removed, the operation being extended above the tentorium in an effort to relieve pressure on the aqueduct. Nevertheless, the patient died shortly after he was returned from the operating theater.

Acute ulceration in the esophagus and stomach constituted the sole associated abnormality seen at autopsy. In the skull a yellowish rubbery nodular tumor arose from the junction of the basiocciput and the basisphenoid. It measured approximately 3 by 2 cm. and extended back 1.3 cm., having eroded the dura and penetrated

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Explanation of Figure 1

Fig. 1 (case 2).—*A*, smear preparation stained with eosin–methylene blue. Note the stalk of connective tissue, with large cells, several multinucleate, attached. Cytoplasmic and nuclear inclusions are present. *B*, paraffin section stained by hematoxylin-Van Gieson method. Cytoplasmic and nuclear inclusions are seen. The arrangement of cells in the intercellular substance is suggestive of chondroma. Compare with figure 3.

into the substance of the brain stem. Cephalad, the posterior communicating arteries were considerably stretched by it.

Microscopically the surgical and autopsy specimens are identical. Smears show typical large cells with granular cytoplasm, darkly stained nucleus or nuclei and many cytoplasmic and nuclear inclusion bodies. Most of the features described in detail in case 3 are to be seen (fig. 1*A*).

Routine sections, however, give a different picture (fig. 1*B*). Here the cells appear to be arranged in groups, or clumps. Some are multinucleate cells or syncytia; others are definitely groups of single cells. Some of the cells have nuclear and cytoplasmic inclusions and vacuoles. Between the cells and groups of cells is a pale monogeneous matrix. This intercellular substance varies in quantity; where

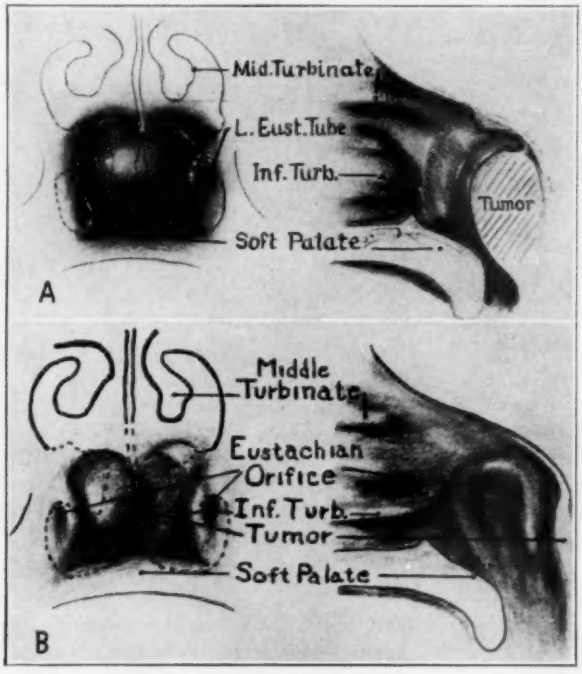

Fig. 2 (case 3).—*A*, drawing of the anterior and lateral views of the nasopharynx as of April 1938. Sketched in outline over the anterior view are the turbinates and septum. The tumor occupies the midline but compresses only the right eustachian tubal orifice. (Drawing by Mrs. Harriet Blockstock Thomson.) *B*, appearance of the same region in January 1940. The tumor is one fifth its previous size and touches but no longer compresses the orifice of the right eustachian tube.

it is abundant, the appearance is strongly enough suggestive of cartilage to explain Virchow's error. Occasional small cystic collections suggest early degeneration.

The sections prove that a thin plate of bone separated the tumor from the mucous membrane of the sphenoid sinus. Where the tumor pushed into the brain stem, the amount of intervening connective tissue was

minimal. The dura, as such, was absent, most of the slight reaction seen being glial. The tumor was an intracranial chordoma.

The following 2 cases represent the group in which the tumor extends into the nasopharynx.

CASE 3.—I. L., a woman aged 50, was admitted to the Montreal Neurological Institute to the service of Dr. W. V. Cone and Dr. McNally.

For two years this patient had had severe headache, radiating into the neck and right shoulder; for nine months she had had double vision; for six months she had noted continuous buzzing in both ears. On first admission (March 1938) there was paralysis of the right lateral rectus muscle and partial loss of function in the right third, seventh, eighth and twelfth cranial nerves. In the nasopharynx, a rounded swelling extended across the vault from the left eustachian cushion to the upper posterior edge of the vomer, filling the nasopharyngeal vault on the right and reducing the right eustachian orifice to a slit (fig. 2*A*). It was firm to palpation, more fibrous than cystic, and tough when grasped with the forceps.

Roentgenograms of the base of the skull showed destruction of portions of the basiocciput, the basisphenoid and the right petrous apex. A soft tissue mass could be seen extending into the nasopharynx.

A punch biopsy specimen contained only lymphoid and fibrous tissue. A specimen from within the tumor was obtained by inserting a 16 gage Luer needle through the tough capsule into the soft tumor beneath. By aspirating with a syringe and moving the end of the needle about within the tumor, blood and fragments of friable gray gelatinoid tissue were obtained. Some bits were smeared between glass slides immediately. The remainder were placed in a 1:10 concentration of a 40 per cent solution of formaldehyde and prepared for section in the routine way.

The microscopic picture of the smear preparation is particularly striking. The tissue was fixed by drying and was stained by Reid's [4] quick frozen section technic. Though some of the cells are distorted, specimens prepared by this method have many features lacking in chemically fixed material. The cells are large, with a distinct cell wall enclosing a finely granular cytoplasm and one or more well stained oval nuclei (figs. 3 and 4). Many cells are attached to a core of connective tissue. Nuclear inclusions are common. One cell in division is seen. In the cytoplasm, particularly in the multinucleate cells, fine pink and yellow granules and large dark bodies, with or without associated vacuoles, are frequently encountered.

Sections of routinely prepared material show more or less well defined rows of large distinct foamy cells, a few of which have multiple nuclei, nuclear inclusions and cytoplasmic bodies. However, none of these features are so strikingly evident as in the smear; preservation of the tumor structure is the sole advantage of this method of preparation.

On the basis of this gross and microscopic pathologic picture, a diagnosis of chordoma was made. Because of its location, the growth was considered inoperable. Therefore six 1 millicurie radon seeds, giving a total of 800 millicurie hours, were planted at equidistant points throughout the mass. Also, roentgen radiation of high voltage was given through four portals over a period of one month, to a total of 4,000 r.

By February 1939 the nasopharyngeal tumor was reduced to one third of its size when first seen. Four more 1 millicurie radon seeds, giving 533 millicurie hours, were inserted. Biopsy of a second specimen taken with needle and syringe, this time through the nostril, showed degenerative changes.

4. Reid, W. L.: Personal communication to the authors, 1938.

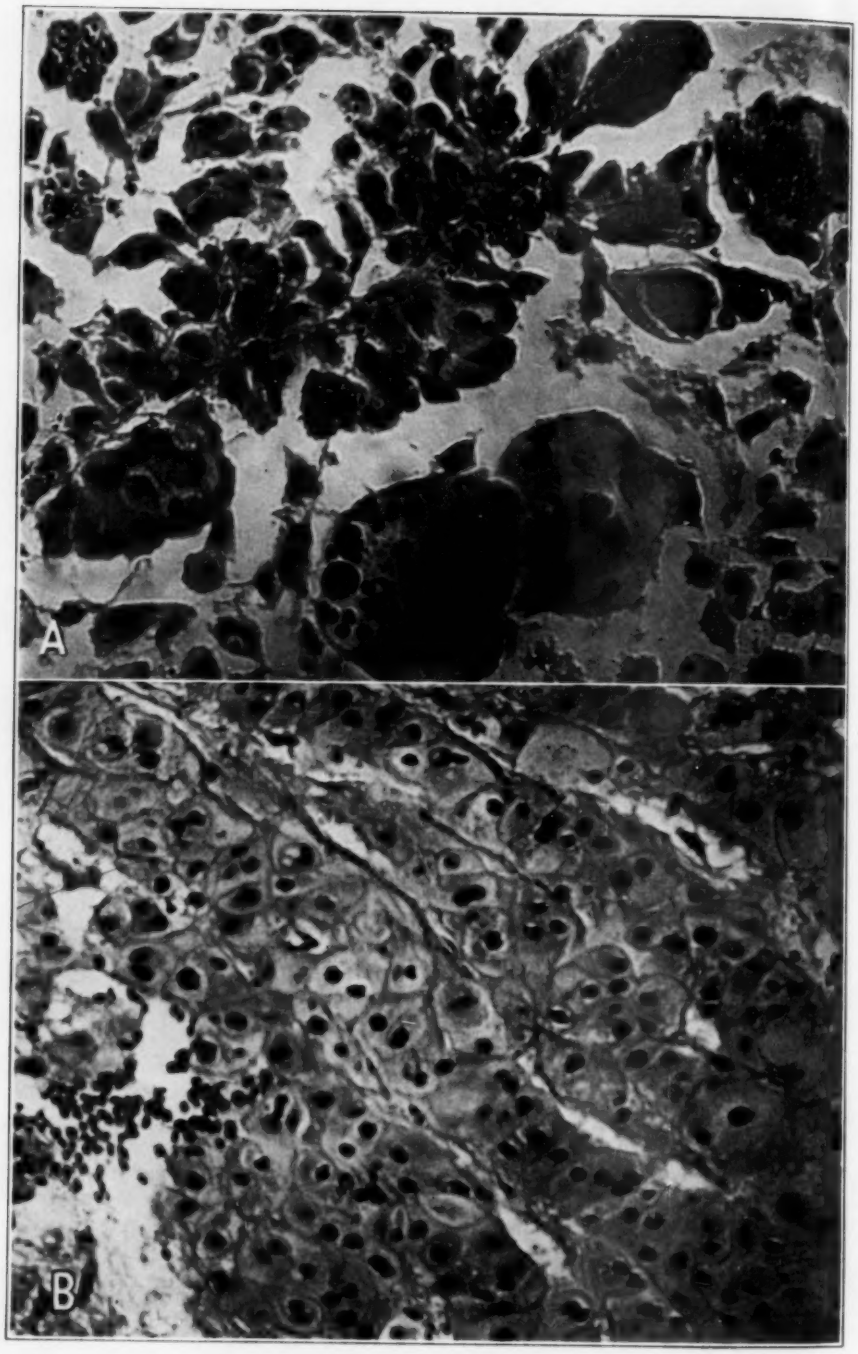

Figure 3

(See legend on opposite page)

In January 1940 the nasopharyngeal projection of the tumor was one fifth of its former size. Confined to the right vault, it touched but did not compress or overlie the right eustachian orifice. It extended down the right posterior nasopharyngeal wall 1.5 cm. from the attachment of the vomer to the nasopharyngeal vault, coming forward 4 to 5 mm. (fig. 2 *B*). The right sixth, ninth, tenth, eleventh and twelfth cranial nerves were almost completely paralyzed. There was evidence of partial involvement of the right third, fifth, seventh and eighth nerves. A constant bilateral noise in the head was still most annoying.

A specimen for biopsy (the third from this patient) was taken through the right nostril by means of a needle. A nasopharyngoscope was in the left nostril, to direct the insertion of the 16 gage needle. The capsule seemed to be much thicker and tougher than previously; the neoplastic tissue was less easily obtained.

In the stained smear preparations there are typical large multinuclear cells with cytoplasmic and nuclear vacuolation. There is not, however, any of the connective tissue stroma noted two years previously. The intracellular bodies brought out so prominently before are not shown. Many cells show various stages of degeneration, even to necrosis. A few clumps of well stained smaller cells containing cytoplasmic vacuoles and occasionally nuclear inclusion bodies are also seen. This indicates neoplastic regeneration in spite of the necrosis produced by the radium emanation.

We are now considering the advisability of exposing the intracranial mass by suboccipital craniotomy and of placing more radon seeds in this portion in which the neoplasm is still advancing. The reduction already obtained seems definitely to indicate such a procedure.

Case 4.—G. G., a girl aged 16, was admitted to the Montreal Neurological Institute to the service of Dr. Cone.

This patient had had a transitory weakness of the right lateral rectus muscle four years before admission. Two years later, a recurrence of this symptom was followed by headache, vomiting, generalized muscular weakness and gradual loss of hearing and vision. There had been marked loss of weight; she had not menstruated in the six month period before admission.

On admission there was complete bilateral loss of function in cranial nerves 2, 4, 6, 7 and 8. The patient obeyed shouted commands. Cranial nerves 3, 9, 10, 11 and 12 appeared to be partially involved. Her appearance was similar to that of a person with Simmonds' disease, though her basal metabolic rate was — 4 per cent.

Roentgenograms of the skull showed almost complete destruction of the sella turcica, the basisphenoid and the basiocciput. Laterally, destruction of bone extended to each foramen spinosum and foramen ovale. The petrous pyramids were eroded as far as the cochleas. A soft tissue mass could be seen in the nasopharynx.

The vault of the nasopharynx was depressed by a smooth swelling in the midline, which extended laterally into the pharyngeal recesses (Rosenmüller's fossae), compressing each eustachian orifice from above and encroaching slightly

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Explanation of Figure 3

Fig. 3 (case 3).—*A*, smear preparation stained with eosin–methylene blue. Some of the large well defined cells are attached to a stalk of connective tissue. The wide variety of cell size, multinuclear arrangement and inclusions in cytoplasm and nuclei can be seen. The varicolored staining of inclusion bodies is lost in the photograph. *B*, paraffin section stained by hematoxylin-Van Gieson method. A few cytoplasmic and nuclear inclusion bodies are seen. The variety of cellular structure seen in *A* is lost, however. Compare with figures 1 and 2.

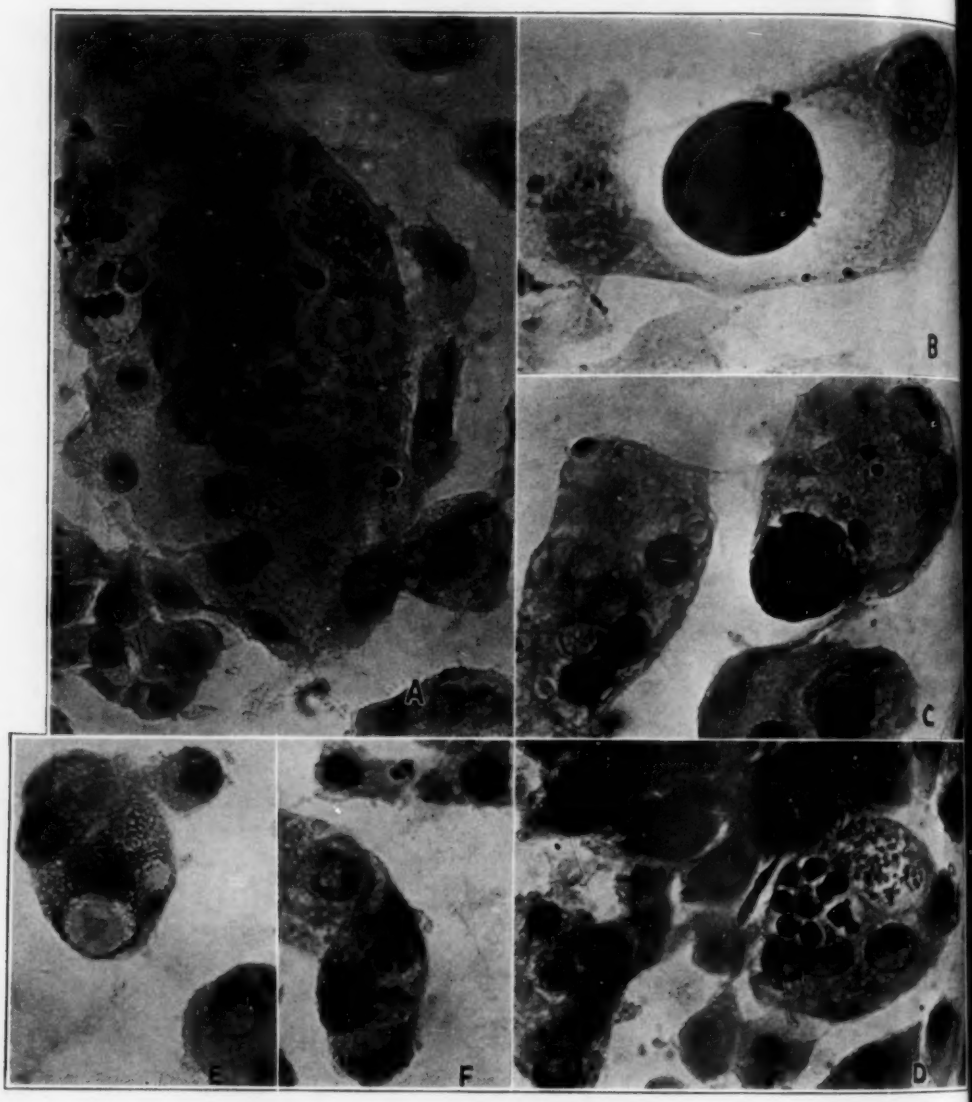

Fig. 4 (case 3).—Smear preparation: *A*, a large multinucleate cell, with a variety of nuclear and cytoplasmic inclusions present. *B*, a cell with a large homogeneous dark-stained body in the cytoplasm. *C*, a similar body breaking up into smaller parts. Other inclusion granules and vacuoles are seen. *D*, a similar body broken up into nine small bodies. Adjacent is a vacuole of small granules. Nuclear inclusions are seen. *E*, vacuoles and granules in a multinucleate cell. *F*, a multinucleate cell with arrangement of material suggesting a stage of mitotic division in a nucleus.

on the attachments of the vomer. The upper portion was firm, but the lower was soft almost to the point of fluctuation. The swelling occupied the normal adenoid area and could be definitely distinguished from the latter only by biopsy. Accordingly, the mass was visualized by a nasopharyngoscope passed through one nostril. A 16 gage needle was introduced through the other nostril, and tissue was taken as in case 3.

The cells found in stained smears of the tumor tissue are large and distinct, are frequently multinucleated and tend to be in grapelike clusters attached to a stem of connective tissue. Nuclear inclusion bodies and cytoplasmic vacuoles are numerous, but the granules seen so graphically in case 3 are not present. The diagnosis was chordoma.

Because of the extent of involvement of the cranial nerves already present, no attempt was made to treat the tumor. Nine months after discharge, the patient's condition is reported to be unchanged.

COMMENT

Diagnosis of chordoma when the tumor is confined to the cranial cavity is difficult. Changes in the basiocciput, basisphenoid and sella turcica have been seen on roentgen examination by others and by us, but in none of our cases was erosion demonstrable in the roentgenograms. Pneumographic visualization has been more reliable and was a major factor in our decision to explore surgically the involved areas in 2 of the patients described here. Even after operation, however, the only finally unassailable diagnostic method was microscopic examination of the tissue removed.

When the tumor projects into the nasopharynx, the diagnosis is made more easily. There is roentgen evidence of erosion of the base of the skull as well as of a soft tissue mass extending into the nasopharynx. This mass can be seen by means of the nasopharyngoscope and can be palpated. We believe that the desirability of a nasopharyngoscopic examination, particularly of the patient with evidence of multiple involvement of the cranial nerves, is to be emphasized. A nasopharyngeal mass must be regarded as possibly sarcoma in an early stage, fibroma or chordoma. All are smooth, round, firm growths which erode the skull, involve cranial nerves and appear in the nasopharyngeal roof. All three may occur in persons of either sex at any age. Fibroma and chordoma are said to be seen more frequently in males—fibroma being commoner in the younger age groups. Fibroma and sarcoma bleed easily; in late stages they may become very large and may develop processes which extend down the nostrils. Therefore, we are again forced to the conclusion that the only certain diagnosis is that made as a result of biopsy.

The all too great hazard of operation is avoidable, for the procuring of tissue by aspiration through a large gage needle, as described in an earlier section of this report, has proved simple and safe and biopsy is conclusive. If it is desired to follow the course of the disease, biopsy can be repeated.

Having established the diagnostic necessity for biopsy, we wish to point out that in our experience smear preparations of chordoma have greater value than cut sections. First, they are more uniform. Figures 1 *B* and 3 *B* are photomicrographs of cut sections, whereas figures 1 *A* and 3 *A* are photomicrographs of smears. There is obviously a greater degree of difference between the first pair than between the second. Besides uniformity, smears have the advantage of speed of preparation. They demonstrate cell form, nuclear content and supporting tissue and preserve within the cell soluble materials ordinarily lost in any of the usual modes of processing. The disadvantage of lost tissue structure and the different types of cellular distortion are adequately compensated for. We have not yet reached the stage where cut sections can be dispensed with. We wish only to emphasize that added information is given by the smear.

The successful removal of a chordoma confined to the cranial cavity has been reported, but the procedure is hazardous. While our results have been disheartening, the indications for operative intervention were absolute in each case, with signs pointing to beginning obstruction of the aqueduct of Sylvius.

In dealing with the nasopharyngeal group of chordomas one's hand is not thus forced. The type of the tumor is safely ascertained. When signs of block appear, operation is an optional procedure. We considered that in one of our patients, the condition was too far advanced to justify any attempt at therapy. The other patient already has shown evidence of great benefit from radium emanation and high voltage roentgen rays. We realize that the chordoma grows so slowly that, even in the absence of any treatment, years may elapse between the time the tumor is identified and the time that it kills its victim. Yet the evidence presented to show the regression of the tumor in case 3 is incontrovertible. It suggests that radium and roentgen therapy should not yet be discarded as valueless for patients with this tumor.

SUMMARY

Chordoma of the basiocciput and basisphenoid may be confined to the cranium and the cranial cavity or may erode into the nasopharynx.

In the latter event biopsy of tissue obtained with a large needle under vision by means of the nasopharyngoscope is simple, safe and conclusive.

Smear preparations of chordoma are quicker to make and more uniform than cut sections and show the true type of tumor better.

Radium and roentgen therapy will produce definite regression of the tumor in some cases and should be given a trial in the treatment of patients with this disease.

Dr. W. V. Cone made many valuable suggestions which have been used in the preparation of this paper.

FINGER EXPLORATION OF THE NASOPHARYNX FOR DIAGNOSIS AND TREATMENT

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In the field of otolaryngology comparatively little emphasis has been placed on the role which pathologic conditions in the nasopharynx play in disturbances of the ear, nose and throat. I refer particularly to the presence of adhesions, adenoids, lymphoid hypertrophy and hyperplasia of the mucous membranes in this area.

Observers have noted the not infrequent occurrence of lymphoid hypertrophy forming ridges on each side of the pharynx or nasopharynx.

The consensus seems to be that some of these excrescences are the result of direct infection of the follicles of the mucous membrane and some are due to drainage from the sinuses.

Several writers have described the excessive lymphoid growth which takes place in young children after the adenoids have been removed. In some cases the presence of these growths after operation may be due to incomplete removal, but there are certainly a considerable number in which after painstaking attempts to remove all the adenoids inspection of the nasopharynx shows that an appreciable amount of lymphoid tissue remains to produce nasal and aural symptoms.

It is undoubtedly true that some children carry these remnants through adolescence and to adult life without the presence of the growths being suspected.

Of these conditions Wright and Smith [1] said:

> They will be found in the nasopharynx with a frequency in exact proportion to the care with which posterior rhinoscopy is carried out, and to the diligence with which the surface is explored by probe and finger.

Barnhill [2] stated that he has frequently noted masses of lymphoid tissue in Rosenmüller's fossae even after supposedly thorough removal of the adenoid tissue and often around the bulbous extremity of the tube.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., New York, June 6, 1940.

1. Wright, J., and Smith, H.: A Text-Book of the Diseases of the Nose and Throat, Philadelphia, Lea & Febiger, 1914, chap. 11, p. 317.

2. Barnhill, J. F.: Personal communication to the author.

Lillie [3] noted:

> The faucial tonsil is the only portion of the tonsillar tissue that can be completely removed, and after its removal there is a tendency in certain cases for the lateral pharyngeal bands and tonsillar plaques in the pharyngeal wall to increase in size to a varying degree. I look on the pharyngeal condition that follows loss of the faucial tonsils as compensatory granular pharyngitis.

During the winter months, when infections of the upper respiratory tract are prevalent, the patient is rare who comes to the rhinologist without presenting some objective and subjective evidence of infection in the nasopharynx and pharynx. It seems that it occurs certainly more often than tonsillitis, which is supposed to be very common. Many of the streptococcic infections or infections of a similar type often have their origin in this area.

If the patient with a nasopharyngeal infection has a temperature higher than normal and shows signs of absorption, prompt rest in bed is the first consideration; next, the application of mild, properly selected medicines; then conservative treatment to the sinuses with some of the mild silver salts. But there often comes a time when these measures fail to clear up the symptoms entirely, and although the patient may feel better generally with the subsidence of acute and subacute symptoms, he is still conscious of some discomfort in the throat. It is often difficult for him to locate and point out the disturbing area. However, when he says that gargles, mouth washes or throat tablets do not reach the point of irritation, one may be quite certain that the involvement is in the nasopharynx. On inspecting this area with the nasopharyngoscope and pharyngeal mirror, the rhinologist can see that the normal pink, smooth and moist membrane is replaced by one which is dull, red, rough and dry. Sometimes definite bands of adhesions can be seen in the fossae of Rosenmüller or in the vault of the pharynx, and sometimes bands from the posterosuperior wall to the lateral walls.

Frequently there may be hyperplasia of the mucous membrane instead of adhesions, or there may be a combination of the two.

For some years it has been interesting to note the influence of abnormalities of the nasopharynx on the proper functioning of the nose and ears. Abnormalities in this area influence the nasal breathing as much sometimes as does a deflection or thickening of the nasal septum of hypertrophy of the turbinates.

Adenoid growths or the remnants of adenoid growths, adhesions and hyperplasia account many times for swelling and obstruction of the eustachian tube, with a resulting loss of hearing of varying degrees. This condition also acts as a retarding influence in the clearing of sinal infec-

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3. Lillie, H. I.: Clinical Significance of Compensatory Granular Pharyngitis, Arch. Otolaryng. **24**:319-324 (Sept.) 1936.

tions. Actually a condition of stasis exists, with its attendant disturbing symptoms. This stasis interferes with one of the important functions of the nasopharynx, which is to act as a passage, or conducting tube, for air and for nasal secretions. The muscles of the pharynx and nasopharynx are almost constantly in motion during the waking hours, because of the acts of talking, eating, drinking and swallowing. This action of the muscles helps to conduct the normal mucus downward and takes care of drainage from the nasal spaces and sinuses. When adhesions or hyperplasias are present these functions are disturbed. Absorption of toxins from the nasopharyngeal area has been described in the literature, and it has been stated by several writers that the constitutional symptoms produced should be recognized.

Brown [4] said:

These lymph follicles act as a station or reservoir, harboring infection for metastasis to other tissues in the body. These lymph follicles are often referred to as a granular pharynx.

Schenck [5] stated:

The high incidence of chronic pharyngeal infection would receive more attention were it not for the fact that the pharynx is apt to be ignored, once the faucial and pharyngeal tonsils have been removed. An appreciable number of patients with obscure conditions attributable to focal infection present no demonstrable focus other than chronic pharyngeal infections. Some patients with unexplained low grade fever regain a normal temperature level after sufficient destruction of abnormal lymphoid nodules in the pharynx.

Crowe [6] made the statement:

The portal of entry for the pyogenic infections of the upper air passages is lymphoid tissue. The hemolytic streptococcus, the pneumococcus and the staphylococcus are the common infecting organisms. In addition to operative removal of tonsils and adenoids we have two other measures that are of value in combatting recurring infection and mechanical obstruction due to hypertrophied lymphoid tissue, i. e., irradiation with radium or Xrays and the administration of sulfanilamide or sulfapyridine during the early stages of acute infection.

Carmack [7] said:

The extreme vascularity of the pharyngeal walls favors rapid absorption of toxic material, and the great number of mucous glands and lymphoid tissue in the pharynx, if involved, tend to increase the toxemia.

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4. Brown, L. E.: The Pharyngeal Lymphatics as a Focus of Infection, Ann. Otol., Rhin. & Laryng. **45**:447-451, 1936.

5. Schenck, H. P.: Chronic Infections in the Pharynx, Arch. Otolaryng. **24**: 299-318 (Sept.) 1936.

6. Crowe, S. J.: The Prevention of Deafness in Children, Laryngoscope **49**: 597-598 (July) 1939.

7. Carmack, J. W.: Conservative Treatment of Pharynx, Ann. Otol., Rhin. & Laryng. **43**:995-1000, 1934.

Drinker [8] and his associates wrote in 1937:

> If the deep cervical lymphatic vessels in the rabbit are cannulated so as to collect the lymph coming from the nasopharyngeal region, one can rapidly recover type III pneumococci which have been instilled gently into the nose.

It is with considerable temerity that I approach the subject of this paper. There is little in the textbooks generally and in the periodical literature that deals with this type of examination. Almost every laryngologist uses finger exploration after adenoidectomy to ascertain whether there are remnants of adenoids. That is practically the extent of finger exploration of the nasopharynx. The sole reliance for diagnosis of conditions in this area is placed on inspection with the nasopharyngoscope and the pharyngeal mirror, both very valuable but not always applicable and not infallible. Finger exploration can give added information and, when indicated, may be used also for treatment.

For almost ten years I have made it a rule with every patient, if the procedure seemed indicated, to explore the nasopharynx with the index finger. The procedure has helped some patients who have been "going the rounds" for relief. Its very simplicity is probably one reason for its being overlooked or not considered. The fact that it is disagreeable is another reason for its not being used. A doctor patient of mine remarked, "It certainly helped me, but don't use it on the occasion of the first examination." With care and practice, however, the procedure can be employed with comparatively little discomfort.

Examination of the nasopharynx is accomplished by the use of the nasopharyngoscope. The regular use of this instrument will often uncover conditions in the postnasal area that might otherwise be overlooked. The orifices of the eustachian tubes, particularly, can be inspected by this method more satisfactorily than by any other examination. It is difficult to use on some children, however, without a general anesthetic.

Avertin with amylene hydrate is used in some clinics for this purpose instead of gas or ether.

Inspection with the pharyngeal mirror is satisfactory in some cases, especially if the pharynx is roomy or if the soft palate does not hug the posterior pharyngeal wall too closely.

A satisfactory examination of the nasopharynx sometimes may be made with the Yankauer pharyngoscope.

Thomson [9] stated that digital examination is an essential procedure in the examination of the nasopharynx.

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8. Drinker, C. K.; Yoffey, J. M., and Sullivan, E. R.: The Absorption of Simple Solutions and Infectious Agents Via Lymph Pathways of the Nasopharynx, Tr. A. Am. Physicians **53**:269, 1938.

9. Thomson, St. C.: Diseases of the Nose and Throat, New York, D. Appleton and Company, 1920, chap. 2, p. 34.

Mithoefer [10] wrote:

> I personally always make it a rule to examine with the nasopharyngoscope as well as with the finger in order to determine the amount of lymphoid tissue present in the fossa of Rosenmüller.

TREATMENT OF THE NASOPHARYNX

Treatment of the nasopharynx includes local medication, the use of sulfanilamide and sulfapyridine (2-[paraaminobenzenesulfonamide]-pyridine), surgical operation, electrocoagulation, irradiation by roentgen rays and radium and finger dissection.

In this paper medication will not be discussed; surgical procedures and electrocoagulation will be only mentioned. Several authorities consider that roentgen rays and radium have their uses in the treatment of the nasopharynx and offer evidence which must be considered valuable. Finger dissection will be discussed, and cases will be presented.

There is no question that irradiation of the nasopharynx by radium and by roentgen rays has a valuable place in the otolaryngologist's armamentarium. Nevertheless, it is not being generally used. The technic of the use of these potent elements must be exact and carefully applied.

Crowe [6] stated:

> The most satisfactory treatment for chronic obstruction of the eustachian tube in children is irradiation with radium or roentgen rays.

Reeves [11] said:

> The lymphoid follicles of the nasopharynx are always predisposing factors in head colds, neuritis and asthmatic bronchitis. There can be little argument against the technique of irradiation of the area by fractional doses of low voltage roentgen rays, as very satisfactory results are obtained after other methods fail.

Eagle and Reeves [12] described the successful treatment of lymphoid hypertrophies and infections in the nasopharynx by roentgen irradiation.

Duncan MacPherson [13] said:

> Lymphoid hypertrophies sometimes respond to radiation therapy but I do not believe it is as successful, judging from my experience, as claimed, although I send my cases to an expert on radiation.

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10. Mithoefer, W.: Pathological Factors Pertaining to Progressive Deafness, Ohio State M. J. **20**:554-555, 1924.

11. Reeves, R. J.: The Radiation Treatment of Hypertrophied Lymphoid Tissue of the Pharynx and Nasopharynx, Am. J. Roentgenol. **37**:510-512, 1937.

12. Eagle, W. W., and Reeves, R. J.: Treatment of Lymphoid Hypertrophies and Infections of Pharynx and Nasopharynx by Irradiation, South. M. J. **29**: 159-163, 1936.

13. MacPherson, D., in discussion on Figi, F. A.: Stenosis of Nasopharynx, Tr. Am. Laryng., Rhin. & Otol. Soc. **36**:176-189, 1929.

Irradiation of diseased faucial tonsils has not been as successful as was hoped. They are an example of infected lymphoid tissue which cannot be eradicated easily.

Schenck [14] expressed a favorable opinion regarding electrocoagulation applied to the affected areas in the nasopharynx. He stated the belief that it is an effective temporary control for the progressive alterations of the lymphoid tissue in chronic pharyngitis.

It is with these thoughts in mind that I venture to suggest that lymphoid tissue may be broken down, ironed out or crushed by finger dissection. This method is not infallible by any means, and when it fails a resort can be made to the other methods mentioned.

Crowe [15] wrote:

> I don't see how massage, breaking up adhesions and flattening out lymphoid hypertrophy, can cause lymphoid tissue to decrease in size.

My only answer is that in many cases the lymphoid tissues and hyperplastic areas are very loosely formed, and for that reason they can be broken up and smoothed out by finger pressure. This ironing-out process, naturally, must be repeated until the tissues firm down.

Friesner [16] said:

> So far as adhesions in the fossa are concerned, I never can see any rationale for breaking these fibrous bands up since they recur rapidly after they have been torn.

It is true that torn adhesions will reform if they are not kept separated, but if the raw surfaces are repeatedly separated they will grow apart in the majority of cases.

Fenton [17] declared:

> I am a strong partisan of palpation of the nasopharynx following adenoid surgery.

Reid [18] wrote:

> When adenoids and tonsils have been removed, many specialists will give the nasopharynx a clean bill of health with little or no examination, even when many symptoms of nasopharyngeal irritation are present; fever, chronic colds, catarrh, rheumatism and obstruction. The attempt is made to explain that situation by deformities in the nose, chronic rhinitis and infection of the sinuses. He (the internist) must not only attempt to look up there with the nasopharyngoscope,

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14. Schenck, H. P.: Chronic Infection of the Pharynx, Pennsylvania M. J. **41**:578-581, 1938.

15. Crowe, S. J.: Personal communication to the author, 1939.

16. Friesner, I.: Personal communication to the author, 1939.

17. Fenton, R. A.: Personal communication to the author, 1939.

18. Reid, C. C.: Naso-Pharynx Pathology After the Adenoid Period, Eye, Ear, Nose & Throat Monthly **17**:153-156, 1938.

but he should insert his finger into the nasopharynx and carefully examine the whole area. Instrumental surgery may be used, but in many cases it is not sufficient. Finger surgery is absolutely essential.

Barnhill [19] stated:

In many instances in my patients where these hyperplasias and adhesions are present the crushing of these tissues with the finger is followed by improvement in hearing and in more normal nasal function.

The following histories give examples of some of the cases in which treatment by finger dissection was used. An effort has been made to eliminate other factors, such as sinusitis and tonsillitis, before attempting the finger operation.

REPORT OF CASES

CASE 1.—About two years ago Mr. H., aged 34. came to me, complaining of difficult nasal breathing and an irritable throat, of which he was always conscious. There was stuffiness in the ears, and they itched constantly. He had seen six rhinologists within the past two years. His tonsils had been removed. Two years previously a submucous resection of the nasal septum had been performed without providing relief. The septum had been reoperated on a year later. The patient said that he still had to breathe through his mouth most of the time and that he was always hawking in an effort to clear the nose and throat passages.

Examination.—The patient had better than normal nasal spacing. The nasopharyngoscope disclosed a small nasopharynx with numerous adhesions, but the true condition was not appreciated until the finger was inserted. The fossae of Rosenmüller were filled with adhesions and hyperplastic soft tissue to the level of the pillars of the eustachian tubes. The anterior salpingopharyngeal fold was similarly affected, and the orifices of the tubes were tight.

Treatment and Progress.—After a thorough finger dissection the bleeding was profuse for several minutes, and the patient said that for the first time in years he could feel the air reach that part of his throat. The finger dissection was repeated at weekly or ten day intervals so that the adhesions would not form again. As a result, the patient has had free nasal breathing for almost two years. He has had fewer colds, and the stuffiness and itching in the ears have disappeared.

Comment.—The benefit which this patient received was outstanding. The location of the firm hyperplasia was such that surgical intervention or diathermic treatment would have been impossible. The use of roentgen radiation or of radium might have helped, but I doubt it, because the hyperplastic lymphoid tissue was more firmly organized than the hypertrophied tissue which Crowe [6] and Baylor treated by radium.

CASE 2.—The patient was a salesman, aged 34. He complained of rheumatic-like symptoms in the neck, shoulders and arms. He had a low grade fever most of the time. He was always conscious of an irritation in his throat, which was hard for him to describe and locate. He had had repeated physical examinations with negative results. He had seen rhinologists in several large cities in his territory.

19. Barnhill, J. F.: Personal communication to the author, 1939.

Examination.—The nares were normal. The tonsils had been removed satisfactorily. The larynx and the sinuses were normal. It was easy to see in the right fossa of Rosenmüller what looked to be a follicle of mucous membrane about the size of a large kernel of corn. From this protruded some caseous material similar to that seen in a tonsil crypt.

Treatment and Progress.—I tried to open the follicle by finger dissection, but it was very resistant. The patient, however, was pleased because, he said, it was the first time any one had touched that area and he was sure it was the seat of his difficulty.

By working on this area persistently at weekly or ten day intervals, I was finally able to open and drain the fossa by finger dissection. The symptoms in the throat began to improve, and the rheumatic symptoms were less marked. The area was then treated every month for about six months, and even after it had healed the patient returned and asked for an examination. The rheumatic symptoms disappeared entirely, and the patient stated that he had never felt better. This case has been followed for about four years, and the nasopharyngeal irritation has not returned.

Case 3.—D. M., aged 24, a husky person with excellent general health, compained of trouble with the nose and throat over a period of two years. He gagged easily on the slightest exertion; there was considerable effort in swallowing but no pain. His nasal breathing was markedly constricted. Smoking, by himself or others, was irritating. It was necessary for him to clear his nose and throat constantly. The tonsils had been removed ten years before. Occasional head colds were severe and tended to persist.

Examination.—When the nose was examined by anterior rhinoscopy and transillumination, the nasal spacing was found to be normal and the sinuses clear. Marked adhesions in the epipharynx and in the fossae of Rosenmüller could readily be seen by means of a pharyngeal mirror. The larynx was normal, but there was marked hypertrophy of the lingual tonsils.

Treatment and Progress.—From the first treatment (Aug. 21, 1938) to the epipharynx by finger dissection the improvement was marked. The nasal breathing improved; postnasal drainage became less and the swallowing easier. On August 29 a second finger dissection was done, and the condition was found to be much improved. There was less bleeding, and the reaction from treatment was only moderate.

The patient has been enthusiastic about the results of manipulation and says the symptoms in his nose and threat have almost disappeared.

Case 4.—D. Q., a boy of 13, was brought to the office because of the following symptoms: more or less constant mouth breathing for about seven years; a hissing sound in the right ear at times for a year; nares constantly filled with thick discharge; recurrent head colds. The tonsils and adenoids had been removed when the patient was 2 years of age.

Examination.—Both nares were practically closed and were filled with mucopurulent discharge. There was some adenoid tissue in the central portion of the nasopharynx and epipharynx but marked hyperplasia and adhesions in Rosenmüller's fossae. The maxillary sinuses were dark on transillumination.

Treatment and Progress.—Irrigation of the antrum gave negative results after several treatments; the nasal spacing improved moderately, and the membranes were not so waterlogged. The improvement was not sufficient, however, to permit of normal nasal breathing. Then the patient was taken to the hospital, and all remnants of adenoid tissue were removed by the LaForce adenotome and

adenoid curets. After this, the nasal condition still improved, but the patient was forced to breathe through his mouth part of the time. From this time on, at weekly or ten day intervals, the nasopharynx was explored by finger. Hyperplastic tissue was broken down and adhesions separated. The improvement in breathing was marked from the beginning. The aural symptoms gradually disappeared, and the nasal membranes became normal in appearance. This procedure was repeated at monthly intervals for about six months.

Recent examination showed that the adhesions had not reformed and the finger massage had so reduced the hyperplastic areas that they seemed to have the same consistency as the surrounding mucous membrane. The patient's father tells me that he is pleased with the results of his son's improvement.

Comment.—It is interesting to note in this case that although there was some improvement after the maxillary sinal infection was cleared up and the adenoids removed, the most marked relief came after treatment to the nasopharynx and epipharynx by persistent finger exploration and massage.

CASE 5.—C. Q., a boy of 10 years, was a brother of the preceding patient. His adenoids had been removed at the age of 18 months, and at the age of 3 years a tonsillectomy was performed and adenoids were again removed.

Regardless of these procedures, the patient breathed through his mouth most of the time. He had recurrent head colds and frequent headaches. During the previous winter the cervical glands had been swollen, and on four different occasions the patient had been put to bed for a week at a time with a sore throat and fever. Cultures were more or less negative. Blood counts showed moderate secondary anemia.

Examination.—The turbinates were pale and soggy; the nasal spacing was poor; the antrums were clear on transillumination and by irrigation. Inspection of the nasopharynx showed marked hyperplasia in the epipharynx and in the fossae of Rosenmüller. There was a small amount of adenoid tissue.

Treatment and Progress.—Finger exploration and massage of the nasopharynx were instituted. From the beginning, improvement in all symptoms has been gradual and permanent. The adhesions have not reformed, and the hyperplastic areas have been thinned down so that they are hardly noticeable.

The boy has had the best summer in years. The headaches and attacks of sore throat have disappeared, and the patient has almost constant nasal breathing.

CASE 6.—M. W., aged 34, complained that she had had a nasal irritation for over twelve years, with restricted nasal breathing most of the time. The tonsils had been operated on twice. The patient had been under treatment at intervals every year with only moderate relief of symptoms and was always conscious of the upper part of her throat. She had the sensation of a chronic cold constantly, with frequent acute exacerbations of the condition. Her personal history, otherwise, revealed nothing of diagnostic significance except that she had had pleurisy and pneumonia in September 1935.

Examination.—The nasal spacing was fairly normal; the maxillary sinuses were moderately dark on transillumination. After three irrigations of the antrums, the haziness disappeared, and there was less irritation in the nose. The breathing was still restricted. There were definite hyperplasia and moderate adhesions in the nasopharynx.

Treatment and Progress.—Finger exploration and massage were begun at this time, and definite improvement resulted. On March 28, 1936, the patient stated that she was feeling better than at any time during the past twelve years. The blunt dissection, or finger exploration, was done again in August 1936 and again in 1937 and 1938. Improvement in symptoms has been maintained, and the patient says she is comfortable.

Comment.—It appears hardly possible that the few treatments described could give so much relief, but this result has been observed in several similar cases, and I believe the facts are worth mentioning.

CASE 7.—A. W. was a girl 19 years of age, in excellent physical condition. Her family physician had examined her repeatedly and could find nothing to account for her persistent cough, husky voice and continued head colds. Her tonsils and adenoids had been removed in 1925. The previous winter she had had nasal and throat treatments for several months without much benefit. The patient was always more or less conscious of irritation in the throat, although it was not actually sore. There was stuffiness of the nose and ears.

Examination.—The nasal spacing was fairly good. On transillumination all sinuses were clear except the left antrum, which was slightly darker. On irrigation, the return solution was foggy, but on two successive occasions it became clear and was clear again a week later. The symptoms did not change much, however. It was difficult to obtain a good view of the nasopharynx by mirror or nasopharyngoscope. Finger palpation disclosed a considerable amount of hyperplastic tissue in the vault and in the fossae of Rosenmüller.

Treatment and Progress.—This soft tissue was broken down by finger massage at weekly intervals. From the first treatment there was definite improvement in symptoms. The huskiness gradually disappeared; the nasal breathing came back to normal, and the stuffiness in the ears was lost entirely. After four treatments to this area the patient was discharged.

Comment.—I have kept in touch with this patient during the past year, and she says she is free from symptoms.

CASE 8.—E. W., a boy aged 10 years, had had recurrent acute head colds and infections of the right middle ear for several years. The tonsils and adenoids had been removed five years previously. Every winter, with each cold, the right ear was sensitive until the nasal symptoms subsided. Several times abscesses had formed and myringotomy had been necessary. The ear would usually drain for two or three weeks.

Examination.—Over a year ago I saw the patient for acute otitis media and bulging of the right ear drum. While the patient was under the anesthetic for myringotomy, the nasopharynx was examined by finger. The left part of the nasopharynx was clear, but on the right side there was some adenoid tissue in the fossa of Rosenmüller and considerable inflammatory and hyperplastic tissue. The nasal spacing was good and the tonsillar area clean.

Treatment and Progress.—On the occasion of the first examination there was free bleeding for a few minutes. The condition of the ear cleared up in about five days. A week later, without anesthesia, finger massage was repeated, and again in about ten days.

The boy's mother tells me that he has had much better nasal breathing. He has had only one or two colds, which lasted three or four days, and they were not accompanied by aural symptoms. He has had the best summer in years.

Comment.—In this case the infected tissue was so located that it would have been difficult to remove with a curet or punch forceps, and then there would have been the probability of adhesions forming.

CASE 9.—D. M., aged 26, complained particularly of more or less constant irritation in the upper part of her throat. Every morning there was a large accumulation of mucus and crusts, which were difficult to dislodge. Nasal breathing had been restricted for several years, especially at night. Two years previously the tonsils and adenoids had been removed, without much improvement in the previously described symptoms.

Examination.—The nasal spacing was rather constricted because of a general thickening of the septum and sogginess of all the turbinates. The membranes were not of the allergic type. There was pus to be seen under both middle turbinates. The pharyngeal mirror showed the epipharynx and the lateral areas to be filled with adhesions and covered with crusts of dried mucopurulent discharge. The large amount of postnasal drainage was suggestive of sinal infection. The antrums were found to be normal.

Treatment and Progress.—The ethmoid region was treated with mild protein silver packs. In about a month the nasal membranes were greatly improved in appearance and spacing was better, but the irritation in the nasopharynx persisted even after the discharge lessened. The adhesions in the nasopharynx were then broken down every week or ten days for about two months. With each treatment, at first, a crust was loosened, which appeared almost like a cast of the nasopharynx. This condition gradually disappeared, and the membranes in this area became smoother; there was practically no bleeding; the dryness was not noticeable, and the nasal breathing became normal.

Comment.—In this case it seemed evident that the infection in the hyperplastic tissue in the nasopharynx was an entity in itself. This tissue probably became infected originally from the sinal drainage, but with prolonged exposure it became a focus itself. Surgical intervention in this area would have been a mistake, I think, because of the inaccessibility of the infected tissue and because of the generalized hyperplasia, and it might easily have produced more adhesions. By persistently following up the finger dissection, I prevented adhesions from reforming, and the other tissues were smoothed out, broken down and crushed so that there were no pockets left to harbor infection.

CASE 10.—Mrs. C., aged 43, was referred to me because of a persistent low grade fever with a temperature ranging between 99 F. and 101 F. The family physician and surgeon were attempting to put the patient in condition for the removal of a large ovarian cyst. They were reluctant to have an anesthetic administered because of the abnormal temperature. Four months previously the patient had had an acute sore throat, but even after the original symptoms had subsided she was still throat conscious.

Examination.—The heart, lungs and urine were normal. A culture from the throat showed hemolytic streptococcic infection, which was not relieved by a course

of sulfanilamide. The sinuses were normal. The tonsils had been removed several years before. Finger exploration of the nasopharynx did not disclose adhesions but disclosed a collection of soft, spongy tissue in the epipharynx.

Treatment and Progress.—This tissue was broken down by fairly firm pressure with the tip of the finger. There was slight bleeding. For the next five days the temperature was normal; then came a slight rise. Finger dissection was done again, and the temperature became practically normal. This treatment was repeated a week later, and the temperature curve became flat.

It has been six months since a hysterectomy was performed. The patient is doing exceptionally well. The throat culture is negative, and there are no symptoms in the throat.

Case 11.—A boy, E. B., aged 15, in good physical condition, complained of difficulty in nasal breathing of six years' duration, worse during the past three years. His nose seemed very tight at times, and when it did open there would be considerable watery drainage. He had constant postnasal drainage, particularly at night. He had occasional attacks of sneezing. He did not complain of headaches or of pressure in the head. He had few head colds, no hay fever, no rheumatism. He did not tire easily. His tonsils had been removed six years previously.

Examination.—The nares were normal; the sinuses appeared normal on transillumination. Irrigations of the maxillary sinuses gave negative results. The tonsillar area was clean. There were marked adhesions and hyperplasia in the nasopharynx, particularly on the right side.

Treatment and Progress.—Finger exploration and dissection were begun immediately and were repeated every week for about two months. From the beginning there was a gradual improvement in the symptoms, and when the patient was discharged, the nasopharynx was much smoother in contour. The low grade redness had disappeared, and there was definitely more room in this area.

Comment.—In this case surgical intervention would have been difficult, as the tissues involved were high in the vault and deep in the fossae of Rosenmüller. There was also the danger of injury to the eustachian tubes and of the production of more adhesions. Blunt dissection with the finger offered a safe and efficient method of dealing with this problem.

Case 12.—Mrs. A., aged 45, was first seen May 12, 1934. Her chief complaint was stuffiness of the nose and irritation, high up in the throat back of the nose and the palate. She had always been subject to severe head colds. She had had arthritis two years previously following a streptococcic infection of the throat, and this condition had cleared up after a tonsillectomy. Her general physical condition was good.

Examination.—The nasal membranes were pale and soggy, with a generalized hypertrophy of the turbinates. The tonsillar area was clean. With the pharyngeal mirror a considerable amount of hyperplastic tissue could be seen in the vault of the nasopharynx and in the fossae of Rosenmüller. The maxillary sinuses were dark and the frontal sinuses clear.

Treatment and Progress.—Irrigations of the antrums disclosed a syrupy mucopurulent discharge. Irrigations were continued at biweekly intervals until the returns were clear. The patient was also given dilute hydrochloric acid three times a day, after meals. The stuffiness of the nose diminished; breathing was easier, and the nasal membranes assumed a more normal color.

Regardless of this improvement, however, the patient still complained of dryness and irritation in the region back of the palate. Finger exploration located the soft tissue and adhesions seen by the mirror. These tissues were ironed out and crushed by rather firm finger pressure every week or ten days for about six weeks. The irritation in the throat gradually became less, and the patient said that for the first time in several years she was not conscious of her throat. The head colds were less frequent, and although there has been a slight return of the irritation in the throat once in a while, each time it has been relieved by finger dissection.

Comment.—I have followed this patient for five years and I, as well as the patient, am convinced that the treatment described has left her almost free from symptoms.

Case 13.—Mr. S., aged 39, was seen in January 1939. He had had a discharge from the right ear since an attack of scarlet fever in early childhood, and treatment of the ear since that time had not reduced the discharge.

Examination.—The patient had subacute bilateral maxillary sinusitis. This cleared up completely in three weeks. The discharge from the ear continued, however. The left eustachian tube was inflated easily, but it was difficult to get any air through the right tube. With the nasopharyngoscope and the pharyngeal mirror, it was seen that Rosenmüller's fossa was filled with engorged hyperplastic tissue; the pillars were thick, and the orifice of the tube was just a slit.

Treatment and Progress.—I used finger dissection in the area of the right fossa and at the eustachian orifice at ten day intervals, on four occasions. Almost from the first dissection the discharge from the ear began to lessen. In about a month it was possible to get some air through the tube, and the ear became dry for the first time in three years. The drum, which was thick and soggy, began to clear and to approach a normal appearance. The hearing also improved.

Case 14.—Miss L., aged 42, was seen in February 1939 because of recurrent head colds since the previous September. A general physical examination gave negative results. Skin tests showed that she was sensitive in varying degrees to about thirty different allergens. She had had a resection of the nasal septum, and the tips of the middle turbinates had been removed, but in spite of these operations and local treatments she could not breathe through the nose. At night she was distressed because of dryness of the throat and aggravation of symptoms when she was lying down.

Examination.—Both nares were completely blocked; the membranes were pale, gray and soggy looking. Transillumination showed the antrums to be dark; the frontal sinuses were moderately dark. Irrigations of the antrums gave positive results. Examination by nasopharyngoscope and pharyngeal mirror disclosed a rather small nasopharynx, with some adhesions in the fossae of Rosenmüller.

Treatment and Progress.—Irrigations of the antrums were repeated every other day, and as the sinuses cleared there was some improvement in breathing, but this was only intermittent. With finger exploration the adhesions in the fossae of Rosenmüller were separated, and the patient for the first time said she felt considerable relief. This procedure was repeated about five times at ten day intervals until the breathing returned to normal. The membranes are still somewhat pale, but the nasal spacing has improved and irrigation of the antrums produces negative results.

Comment.—The important thing to remember in this case is that, notwithstanding the clearing up of the sinal infection, which was necessary and important, the patient obtained more definite relief from aeration of the nasopharynx.

SUMMARY

The occurrence of adhesions, hyperplasias and collections of lymphoid tissue in the nasopharynx is much more prevalent than is generally considered.

With care and gentleness, the examining finger may often be used to diagnose conditions in the nasopharynx when the nasopharyngoscope and the pharyngeal mirror fail to give a satisfactory estimate. This exploration may be employed as a method of treatment as well as a means of diagnosis.

When finger exploration and dissection are indicated, the results are immediate and definite. In a long series of examinations I have yet to see any harm from this procedure. The very simplicity of this examination may be one reason why it has not been more generally applied.

Finger exploration of the nasopharynx by its very nature has its limitations, but used carefully and sanely it is applicable to several common pathologic conditions.

For the treatment of hyperplasias in the vault of the epipharynx or in the fossae of Rosenmüller, I believe that this method is more practical and safer than surgical intervention or electrocoagulation and that it is a valuable complement to irradiation by roentgen rays and radium.

SULFATHIAZOLE FOR TREATMENT OF STAPHYLOCOCCIC MENINGITIS

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Sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole), the newest of the sulfanilamide group of drugs, is ready to take its place in the armamentarium of chemotherapy. Sulfathiazole therapy has progressed so rapidly since the release of the drug that the literature associated with it is poorly organized. Our interest in the drug was stimulated by what impressed us as an almost unbelievable therapeutic result in what we had feared was an incurable condition. The report of this case follows. We feel that a preliminary review of the literature concerning sulfathiazole is worth while at this time.

Sulfathiazole is the thiazole analogue of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) and was first reported by Forsbinder and Walter [1] and by Lott and Bergeim.[2] These authors described the methods of preparation and their experiments with these drugs against streptococcic and pneumococcic infections in mice. The original work on the physical and chemical characteristics of sulfathiazole and its salt was done at that time, and the tests for the estimation of the drug in the blood were described.

Van Dyke [3] and his co-workers showed the rapid metabolism of this drug in the body. Sulfathiazole was proved to be more rapidly absorbed than the other sulfanilamide derivatives, the blood concentration reaching its highest level in one to three hours after ingestion. Further, it dis-

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1. Forsbinder, R. J., and Walter, L. A.: Sulfanilamide Derivatives of Heterocyclic Amines, J. Am. Chem. Soc. **61**:2032 (Aug.) 1939.

2. Lott, W. A., and Bergeim, F. H.: 2(P Amino-Benzenesulfonamido) Thiazole: A New Chemotherapeutic Agent, J. Am. Chem. Soc. **61**:3593 (Dec.) 1939.

3. Van Dyke, H. B.; Greep, R. O.; Rake, G., and McKee, C. M.: Observations on the Toxicology of Sulfathiazole and Sulfapyridine, Proc. Soc. Exper. Biol. & Med. **42**:410 (Nov.) 1939.

appeared and was excreted more rapidly. These results led to the conclusion that this drug must therefore be less toxic than the others.[4]

Reinhold, Flippin and Schwartz [5] contributed material on the disposition and action of the drug in man from the standpoints of pharmacology and toxicology. Their conclusions can be summarized as follows:

1. Sulfathiazole is rapidly absorbed from the gastrointestinal tract and rapidly excreted in the urine.
2. Intravenous administration of the sodium salt results in almost complete quantitative excretion in the urine in twenty-four hours.
3. The toxic effects are:
   (*a*) Transitory oliguria.
   (*b*) Transitory and mild dysfunctions of the hemopoietic system.
   (*c*) Mild and only infrequent vomiting.
   (*d*) In a few cases (less than 3 per cent), dermatitis and psychoses.

In general, toxic reactions from sulfathiazole are rare and mild. However, in a few cases hematuria [6] and peripheral neuritis [7] have been reported.

This drug may be applied to the same types of infection and bacteria as the other derivatives and has been shown by many authors to be superior to sulfanilamide in most cases.[8] Because the causative organism in our case was Staphylococcus, we were particularly interested in the unusual effectiveness of sulfathiazole against that organism. The literature is full of reports which bear out our observations. Rake and McKee [8c] reported studies made in vitro and in mice to determine the action of sulfathiazole, sulfapyridine and sulfamethylthiazole (2-sulfanil-

4. Long, P. H.; Haviland, J. W., and Edwards, L. B.: Acute Toxicity, Absorption and Excretion of Sulfathiazole and Certain of Its Derivatives, Proc. Soc. Exper. Biol. & Med. **43**:328 (Feb.) 1940.

5. Reinhold, J. D.; Flippin, H. F., and Schwartz, L.: Observations on the Pharmacology and Toxicology of Sulfathiazole in Man, Am. J. M. Sc. **199**:393 (March) 1940.

6. Arnett, J. H.: Hematuria from Sulfathiazole Therapy in Pneumonia, J. A. M. A. **115**:362 (Aug. 3) 1940.

7. Northey, E. H.: Structure and Chemotherapeutic Activities of Sulfanilamide Derivatives, Chem. Rev. **27**:85 (Aug.) 1940.

8. (*a*) McKee, C. M.; Rake, G.; Greep, R. O., and Van Dyke, H. B.: Therapeutic Effects of Sulfathiazole and Sulfapyridine, Proc. Soc. Exper. Biol. & Med. **42**:417 (Nov.) 1939. (*b*) Cooper, F. B.; Gross, P., and Lewis, M.: Chemotherapeutic Evaluation of Sulfanilamide Derivatives of Heterocyclic Amines, ibid. **42**:421 (Nov.) 1939. (*c*) Rake, G., and McKee, C. M.: Action of Sulfathiazole and Sulfamethylthiazole on Staphylococcus Aureus, ibid. **45**:561 (March) 1940. (*d*) Bliss, E. A., and Ott, E.: Effects of Sulfapyridine, Sulfathiazole and Sulfamethylthiazole upon Severe Staphylococcus Infections in Mice, ibid. **43**:706 (Feb.) 1940.

amido-4-methylthiazole) on Staphylococcus aureus. In both series of experiments, the first-named drug was shown to be superior. Other authors [9] showed experimentally that sulfathiazole is an effective bacteriostatic and bactericidal agent against staphylococci and much superior to sulfanilamide, sulfapyridine or sulfamethylthiazole. In actual staphylococcic infection (septicemia,[10] wound infections [11] and meningitis [12]) the greater prophylactic and curative effect of sulfathiazole has been demonstrated. An interesting observation by Browning [13] on the use of staphylococcus antitoxin in conjunction with the drug led us to combine the two in our case. Browning concluded that staphylococcus antitoxin when given prophylactically has some protective action and may increase the curative effect of sulfathiazole.

The suggested dosage has varied with many observers. We followed the suggestions of Reinhold [5] and his associates, that the best method of administration is the continued one, i. e., 3 Gm. initially and 1 Gm. every four hours. In this way, the concentration will vary from 1.2 to 19 mg. per hundred cubic centimeters of blood, with free sulfathiazole averaging about 5.45 mg. per hundred cubic centimeters of blood.

One contradictory observation is noteworthy. Sulfathiazole has not been found in any appreciable concentration in the spinal fluid. The mechanism of its action in our case and in the cases of others [12] must therefore be more thoroughly investigated. That the action is specific is undoubted by us, since we can show a definite relationship between the spinal fluid findings and the sulfathiazole dosage in our case.

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9. Bliss and Ott.[8d] Barlow, O. W., and Homburger, E.: Specific Chemotherapy of Experimental Infections with Thiazol Derivatives of Sulfanilamide, Proc. Soc. Exper. Biol. & Med. **42**:792 (Dec.) 1939. Lawrence, C. A.: Bacteriostatic Action of the Three Thiazol Derivatives of Sulfanilamide upon Bacteria in Broth Culture, ibid. **43**:92 (Jan.) 1940. Rammelkamp, C. H., and Keefer, C. S.: Sulfathiazole: Effects on Staphylococcus Aureus in Vitro, ibid. **43**:664 (Dec.) 1939.

10. Herrell, W. E., and Brown, A. E.: The Clinical Use of Sulfamethylthiazole in Infections Caused by Staphylococcus Aureus, Proc. Staff Meet., Mayo Clin. **14**:753 (Nov. 29) 1939. Stirling, W. C.: Sulfathiazole: Two Cases of Septicemia with Recovery, J. A. M. A. **115**:118 (July 13) 1940.

11. Herrell, W. E., and Brown, A. E.: Local Use of Sulfamido Compounds in the Treatment of Infected Wounds, Proc. Staff Meet., Mayo Clin. **15**:611 (Sept. 25) 1940.

12. Dietel, F. W., and Kaiser, A. D.: Staphylococcus Aureus Meningitis Successfully Treated with Sulfathiazole, J. A. M. A. **115**:601 (Aug. 24) 1924.

13. Browning, P.: The Chemotherapy of Experimental Staphylococcus Infections in Mice with Drugs of Sulfonamide Group, J. Path. & Bact. **50**:431 (May) 1940.

REPORT OF A CASE

On Aug. 6, 1940 a 29 year old man of Italian parentage entered the Illinois Eye and Ear Infirmary in a stuporous condition. The history obtained was that two weeks previously otalgia had developed on the right side. This had been treated by myringotomy. The aural discharge that had followed was scanty and of short duration. Two days prior to his admission, severe headache, mainly localized to the right temporal and occipital regions, had developed. His condition grew progressively worse, and on the night before hospitalization pain and stiffness of the neck developed and he gradually lapsed into a stuporous state.

Examination on admission revealed a stuporous, nonresponsive, mumbling patient with both neck and back in semiextension and markedly rigid. The temperature was 102 F., the pulse rate, 96 and the respiratory rate, 52. The blood pressure was 160 systolic and 104 diastolic. There were extreme tenderness over the right mastoid and changes in the right drum membrane, which appeared thick and had no visible landmarks and no light reflex. The posterior half of the membrane was deeply injected and bulging in the superior quadrant. Functional tests were inconclusive because of the patient's unresponsive state. Other examinations gave essentially negative results except for a positive Kernig and a right Babinski sign. Examination of the blood showed the hemoglobin content 91 per cent, the red blood cells 4,900,000 and the white blood cells 25,000, with 91 per cent polymorphonuclears. The urine showed albumin (2 plus) and on microscopic examination a few white and red cells were found. Spinal tap was done with extreme difficulty because of the opisthotonos. The fluid was cloudy and apparently under increased pressure; it contained 5,500 cells, predominantly polymorphonuclear. Chemical analysis showed globulins 4 plus, total proteins 1.2 per cent, chlorides 681 mg. per hundred cubic centimeters and sugar 102 mg. Roentgenograms showed definite involvement and breakdown of the entire cell structure of the right mastoid (pneumaticized).

A diagnosis of otitic meningitis was made and surgical intervention advised. A simple complete mastoidectomy was performed on the right by the postauricular route, and all the mastoid cells were found to be broken down and to contain a greenish gray necrotic material and some frank creamy pus. Complete exenteration was performed. The dura of the middle fossa was exposed and found to be covered with granulations; the lateral sinus was exposed and found to be of a gray color; it bled freely on incision.

After the operation, the administration of sulfanilamide was started with an initial dose of 30 grains (1.94 Gm.), repeated in two hours and then followed by 20 grains (1.29 Gm.) every four hours day and night. Thereafter the course was stormy in spite of repeated blood transfusions and a dosage of sulfanilamide up to 150 grains (9.7 Gm.) daily, by mouth and parenterally. The blood level of sulfanilamide was maintained at 10 mg. per hundred cubic centimeters, but the patient showed little response, the temperature remaining high and the signs of meningitis increasing. On the third postoperative day, evidence of definite pulmonary congestion was found, and this, in addition to the absence of response of the patient to the drug being used, led us to stop giving it and commence using sulfapyridine, a total dose of 120 grains (7.8 Gm.) being given daily. A slight improvement was noted, the temperature becoming gradually lower, the nuchal rigidity diminishing and the patient becoming rational and responsive for the first time since admission to the hospital. Unfortunately, frank hematuria developed, and we were forced to stop the administration of sulfapyridine, since we felt that the

complication was probably due to acetylsulfapyridine crystals. Fluids were forced, and the administration of sulfanilamide was resumed.

During the next three or four days there was a gradual rise in temperature, and finally the severe headache and all the signs of meningitis recurred. After this there was a gradual but definite loss of ground, with every sign of a toxic process. The blood picture again took on its original appearance, and the temperature fluctuated daily from 100.4 to 105 F. Each succeeding spinal puncture showed an increase in the cell count, and the patient again became stuporous. On the fifteenth postoperative day he appeared moribund. The pupils were unequal and nystagmus developed, but hearing was still retained in the affected ear. At this time the first positive report on the cultures of the spinal fluid was returned. The fluid showed a growth of hemolytic Staphylococcus albus. On the basis of the preliminary report, it was decided that nothing would be lost in trying the new sulfanilamide derivative, sulfathiazole. Since the condition of the patient appeared critical, the sodium salt of sulfathiazole was given intravenously in a 5 per cent solution (5 Gm. in 100 cc. of triple distilled water). This was repeated at intervals of seven hours until three doses, or 15 Gm. (225 grains), had been given. Overnight—August 23-24—there was a very great improvement, with the temperature becoming normal in less than twenty-four hours and all the symptoms of meningitis abating. Because we were unfamiliar with this new drug, which was considered dangerous, we stopped its administration and resumed that of sulfapyridine.

On the twenty-second postoperative day there was a sudden recurrence of headache and of fever, which reached 102 F. The next day a report on the blood culture finally revealed Staph. albus as the causative factor of the septicemia. The administration of sulfathiazole was resumed, the dosage being three tablets (7.7 grains [0.52 Gm.] each) given by mouth every four hours day and night. A spectacular improvement occurred within sixteen hours. The headache disappeared, and the temperature dropped to normal. This improvement persisted during the administration of the drug. A few days later the drug became unavailable. Within twelve hours after the last dose of sulfathiazole the patient's temperature again rose and the severe headache returned. A new supply of the drug was obtained, and a short while after readministration, the symptoms subsided and the temperature became normal.

On the thirtieth postoperative day the patient seemed normal and happy. Findings were at a minimum. The spinal fluid cell count was down to less than 100 cells. We decided to test the withdrawal of sulfathiazole. About forty hours after the last dose of the drug the patient was moaning loudly, calling for help and occasionally lapsing into a semistupor. His temperature rose to 102 F. When the administration of the drug was resumed, amelioration of the symptoms was much slower than on previous occasions. The frequent reccurrence of meningitic symptoms whenever the patient did not receive sulfathiazole suggested the presence of a small localized area of infection, which undoubtedly fed the general meningitis. It was considered dangerous to continue such heavy dosage of sulfathiazole (135 grains [8 Gm.] daily) much longer, and on the forty-second postoperative day the patient was given 100,000 units of polyvalent staphylococcus antitoxin in divided doses over a period of two hours. The dosage of sulfathiazole was gradually diminished, and there was a steady and permanent improvement, persisting after complete withdrawal of the drug. The patient was discharged cured sixty-five days after admittance and has had no further symptoms to date.

CONCLUSIONS

1. Sulfathiazole is rapidly absorbed and metabolized and equally rapidly excreted from the body.

2. Its toxicity is low, and severe complications from its use are infrequent.

3. Its action on Staphylococcus is somewhat specific, and in spite of its failure to reach the spinal fluid, the drug is extremely beneficial in the treatment of staphylococcic meningitis.

4. Staphylococcus antitoxin when used in conjunction with the drug is of considerable value.

25 East Washington Street.

Case Reports

OSTEOMA EBURNEUM OF THE MASTOID

ALBERT A. CINELLI, M.D., NEW YORK

Osteoma of the mastoid is so rare that only recently has this subject been given some clarification. Proof of its rarity is the fact that only 28 cases have been reported in all medical literature.

Osteoma of the mastoid should not be confused, as is often done, with exostosis of the external auditory canal. The latter condition invariably arises from the tympanic portion of the temporal bone, and this anatomic situation alone distinguishes it from osteoma of the mastoid process.[1]

The otolaryngologist usually sees osteoma in the lumen of the frontal sinus or in the ethmoid capsule, but rarely in the mastoid region. When the growth is found in the mastoid region it can be classified as a true osteoma rather than as an exostosis or a hyperostosis.[2]

Histologically there are three types of osteoma of the mastoid:

1. Osteoma compactum, or osteoma eburneum, occurs more frequently than the other types. It is hard, like ivory, spherical; it is attached to the cortex of the mastoid and may even penetrate into the mastoid cells. Histologically it shows dense, lamellated bone.

2. Osteoma cancellare is exceedingly rare. Only one case has been reported, that of Geschickter and Copeland.[3] Histologically it consists of cancellous bone, plus fibrous cellular tissue.

3. Osteoma cartilagineum consists of bone and cartilage. This anomaly is not uncommon and has been reported by many writers.

Clinically it may be difficult to distinguish the type of osteoma because of the similarity of the symptoms and objective signs observed in connection with the various types. The differential diagnosis depends chiefly on the histologic examination.

The causation is unknown. According to Haymann,[4] the tumor develops after the age of puberty and is dependent on the condition of growth in the cranial bones. Preusse[5] seemed inclined to think that the constitutional and glandular condition is the cause.

1. Cheatle, A. H.: Some Points on the Surgical Anatomy of the Temporal Bone from Birth to Adult Life, London, J. & A. Churchill, 1907, p. 42.

2. Coates, G. M.: Osteoma Growing from the Mastoid Cortex, Arch. Otolaryng. **28**:27 (July) 1938.

3. Geschickter, C. F., and Copeland, M. M.: Tumors of Bone, New York, American Journal of Cancer, 1931, p. 19.

4. Haymann, D.: Zur Kenntnis der Knochengeschwülste des Warzenfortsatzes, Ztschr. f. Ohrenh. **78**:23, 1919.

5. Preusse, K. H.: Über Osteombildung am Warzenfortsatz Felsenbeins, Beitr. z. prakt. u. theoret. Hals-, Nasen- u. Ohrenh. **31**:203, 1934.

Stuart [6] suggested that a pituitary dysfunction might influence this condition. The patient in the case which he presented did not show any evidence of dyspituitarism, but her father and elder brother had acromegalic features and two of her sisters showed a tendency toward atrophic rhinitis, a condition which has recently been reported associated with disturbances of the pituitary gland.

Friedberg [7] suggested that trauma, with subsequent ossifying periostitis, and chronic inflammation may be predisposing agents. As in the case of most neoplasms, no definite cause can be ascertained.

Osteoma may cause no disturbance whatever, or it may present definite pressure symptoms. There may be pain referred to the ear, or drum, or to the neck, due to irritation of the greater auricular and the small occipital nerves. The tumor may infiltrate the cortex well

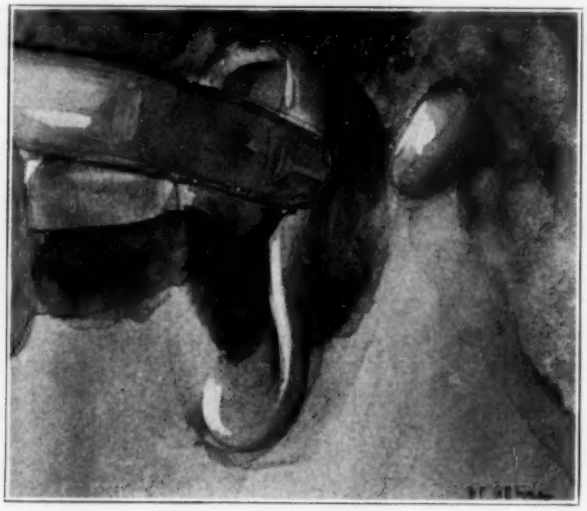

Fig. 1.—Illustration showing the size and position of the osteoma.

forward, causing the posterior meatal wall to be pushed forward and downward, thereby producing deafness of the conduction type.

An interesting fact to observe is that these tumors usually occur in woman beyond the age of puberty.

The treatment for all types of osteoma is surgical. After careful removal of the tumor, all symptons usually disappear.

REPORT OF A CASE

A white married woman, 33 years old, was admitted Feb. 24, 1940, to the Manhattan Eye, Ear and Throat Hospital (Dr. Buckley's clinic) because of a hump behind the left ear. This mass had been progressively growing for the past two years. There was no history of any trauma. The patient complained that she

6. Stuart, E. H.: Osteoma of the Mastoid, Arch. Otolaryng. **31**:838 (May) 1940.

7. Friedberg, S. A.: Osteoma of the Mastoid Process, Arch. Otolaryng. **28**: 20 (July) 1938.

had suffered from severe headaches during the past year and from intermittent attacks of dizziness during the past two weeks. There was present in the left ear and extending down into the neck some pain which had started several weeks before and was becoming worse.

Family History.—The family history was essentially noncontributory, except that the mother died of carcinoma of the breast.

Examination.—The patient was a young woman of healthy appearance. Behind the left ear was a mass about 2.5 cm. in diameter, fixed to the mastoid cortex (fig. 1). It was firm, smooth and hard to the touch. The skin was fully movable over it. Deep pressure over the mass elicited tenderness and some pain to the left ear and down into the neck.

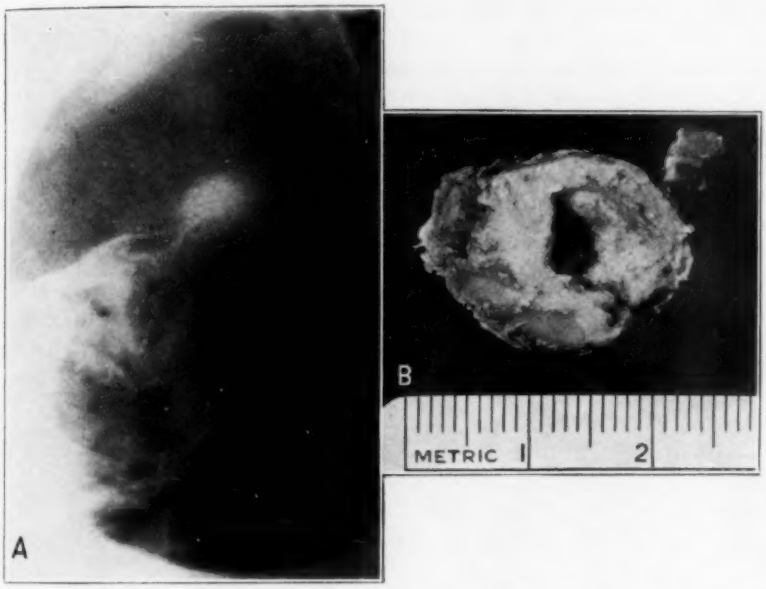

Fig. 2.—*A*, roentgenogram of the osteoma; *B*, photograph of the gross specimen of the osteoma removed.

The nose and throat and the tympanic membranes were normal. Audiometric and vestibular tests gave essentially negative results.

The heart, lungs and abdomen were essentially normal. Neurologic examination indicated no significant deviation from the normal. A pathologic condition of the endocrine system was not discernible. Complete examination of the blood, urinalysis and determination of the blood calcium revealed nothing abnormal. The Wassermann test was negative. The roentgenogram, made by Dr. Frederick M. Law, showed an osteoma, the size of a large marble, at the knee of the sinus but not involving the inner table (fig. 2 *A*).

Operation and Later Course.—The patient was admitted March 5, 1940, for surgical removal of the osteoma. Nitrogen monoxide–ether anesthetization was started at 2:25 p. m. and terminated at 3 p. m. The patient was prepared and draped as for a mastoidectomy. A postauricular incision was made over the left mastoid, and the skin and periosteum were retracted. A smooth, firm, fixed mass, about 2.5 cm. in diameter, was located over the upper region (fig. 2 *B*).

The osteoma, which infiltrated the cortex of the mastoid but did not involve the mastoid cells, was removed with a chisel. The edges of the skin were approximated with Michel clamps, and no drain was inserted.

The patient made an uneventful recovery and on March 11, 1940, was discharged in good condition. When last seen in the clinic, she said the pain and dizziness had left her completely.

Pathologic Examination and Diagnosis.—The microscopic report of the biopsy, by Dr. Andrew A. Eggston, revealed that the specimen showed rather dense lamellated bone and distinct haversian canals with blood vessels. The lamellae were arranged concentrically around the canals. There was some intervening bone of a granular type, and a small bit of intratrabecular connective tissue. In some areas there were strands of calcium salt deposits without bone formation. There was little inflammatory reaction. The haversian systems were well outlined and numerous, as many as ten or twelve per low power microscopic field.

The diagnosis was osteoma of the lamellated bone type.

SUMMARY

A cursory review of the classification and etiology of osteomas of the mastoid bone is given. A case in which osteoma eburneum mastoideum, of unknown cause, was associated with pain and some dizziness is reported. Complete relief from all symptoms was obtained by surgical removal of the tumor.

1021 Park Avenue.

OSTEOMYELITIS OF THE FRONTAL BONE IN THE ABSENCE OF FRONTAL SINUSES

ROBERT B. LEWY, M.D., CHICAGO

Adequate and accurate prognostic knowledge in handling osteomyelitis of the frontal bone is still wanting. The histologic studies of Mosher and others were a step forward. Yet, today's successes in the treatment of this condition are so frequently punctuated by failure that further study seems justifiable. New or unusual etiologic factors which seem to confuse today's picture may be a key to tomorrow's solution of the problem.

At present, the cases reported indicate that the process is an extension of frontal sinusitis to regions where the tissue is not equal, in local and general resistance, to the task of walling off the infection. Pastore and Williams [1] presented a case in which the disease progressed from an active frontal sinusitis to an extradural abscess. Extensive frontoethmoidectomy controlled the disease. Adson and Hempstead [2] presented cases in which frontal sinusitis was a direct vector. They stated that there are three types of osteomyelitis of the frontal bone—fulminating, localizing and spreading, each with its own prognosis. The principles of treatment include adequate drainage of the sinus with removal of the necrotic bone, preservation of the periosteum and the confinement of scars to the area within the hair line. In 1930 Woodward [3] stated that osteomyelitis of the frontal bone as a complication of frontal sinusitis is rare and that no one had reported more than 3 cases. In 1938 McKinney [4] reported 8 cases and stated that frontal sinusitis preceded osteomyelitis in the majority of these. There was no evidence in any of his cases that the osseous infection did not follow frontal sinusitis. Frontal sinusitis preceded the disease of the frontal bone in the case reported by Weiss.[5]

Read before the Chicago Otolaryngological Society, March 4, 1940.

From the Department of Otolaryngology, University of Illinois College of Medicine.

1. Pastore, P. N., and Williams, H. L.: Osteomyelitis of Frontal Bone Secondary to Suppurative Disease in Frontal Sinus: Report of Cases, Proc. Staff Meet., Mayo Clin. **13**:7 (Jan. 5) 1938.

2. Adson, A. W., and Hempstead, B. E.: Osteomyelitis of Frontal Bone Resulting from Extension of Suppuration of Frontal Sinus: Surgical Treatment, Arch. Otolaryng. **25**:363 (April) 1937.

3. Woodward, F. D.: Osteomyelitis of Skull: Report of Cases Occurring as Result of Frontal Sinus Infection with Staphylococcus Pyogenes-Aureus, J. A. M. A. **95**:927 (Sept. 27) 1930.

4. McKinney, R.: Osteomyelitis of Frontal Bone: Report of Eight Cases, Arch. Otolaryng. **28**:1 (July) 1938.

5. Weiss, J. A.: Circumscribed Perforative Osteomyelitis of Frontal Sinus, Illinois M. J. **74**:169 (Aug.) 1938.

Sometimes, of course, osteomyelitis follows trauma or some type of surgical intervention, but the common expression of its development is the sequence of occlusion of the ostium, pus under pressure, consequent diminution of the blood supply and death of tissue. This necrosed tissue, an ideal culture medium, is then kept at incubation temperature, and the rest of the history is inevitable.

Two cases are here presented: one is that of a child, the other that of an adult, in whom osteomyelitis developed despite the absence of frontal sinuses.

REPORT OF CASES

CASE 1.—M. W., a woman aged 44, noticed in June 1937 a general malaise and a loss of appetite and weight. General examination at that time, including roentgenograms of the sinuses, gave negative results. During the succeeding month she did not improve, and by July there was a marked swelling about the left eye. At this time the left maxillary sinus was irrigated twice a week. After a brief

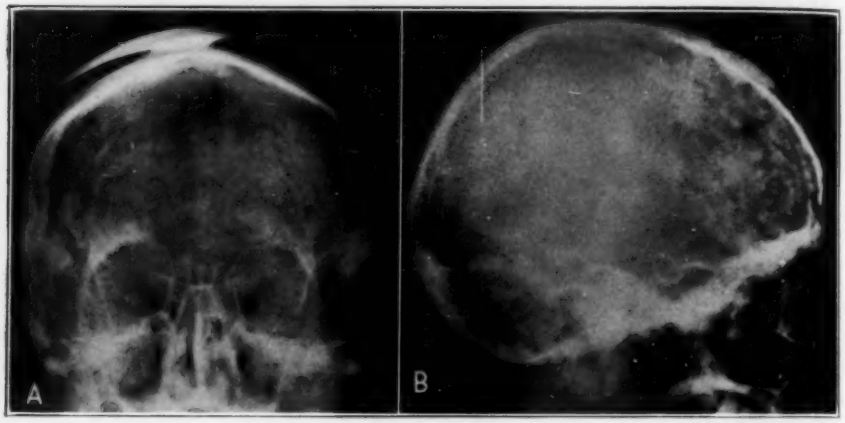

Fig. 1.—Roentgenograms of M. W.: *A*, anteroposterior view, demonstrating marked osteomyelitis of the frontal bones and of the calvarium and absence of frontal sinuses. *B*, lateral view, showing the osteomyelitic process and the absence of frontal sinuses.

period of apparent recovery, the lower eyelid began to swell and the swelling closed both lids. The sinus was irrigated again, and three days after this the area of the face below the left eye swelled and became very painful.

Physical examination on admission revealed the left eye completely closed, with marked swelling beneath. This area, with part of the lateral wall of the nose, was hot, red and tender. Diagnosis at this time was acute maxillary sinusitis on the left side.

Laboratory examination showed acetone, 2 plus, and diacetic acid in the urine. Sugar was found on one occasion. The white blood cells numbered 14,750 and the red blood cells 3,740,000 per cubic millimeter. The value for hemoglobin was 65 per cent. The differential count was: polymorphonuclears, 81 per cent; eosinophils, 1 per cent; lymphocytes, 12 per cent; monocytes, 4 per cent and irritation forms, 1 per cent.

Roentgen Examination of the Sinuses.—In the first roentgenogram, taken on September 10, the left antrum appeared dense and there was seen to be a loss of substance on the lateral side, due either to operative work or to necrosis of the bone. The frontal sinus was congenitally absent except possibly for a small central rudimentary cell. On October 22, forty days later, there was a distinct involvement in the frontal osseous area, which was mottled and pathognomonic of osteomyelitis. The central area of the left antrum had cleared, but there was more destruction of bone on the lateral aspect, where it joined the malar bone and the zygoma. The subsequent roentgenograms showed even more extensive involvement. A pathologic examination by Dr. R. H. Jaffé on November 2 showed chronic inflammatory changes, with evidence of recent and old hemorrhages of the frontal bone; there was acute suppurative osteomyelitis with active infection in some parts of the bone marrow and fibrosis in other parts. A culture revealed Streptococcus haemolyticus.

Surgical Procedures.—Operation was performed on September 14 by the sublabial approach. The facial surface of the maxilla was removed. A large opening was made under the inferior turbinate. A rubber drain was left in place. A septal abscess developed and was drained. On October 26, on account of extension of the process to the roof of the orbit and the crista galli, a portion of the frontal bone, extending 1½ inches (3.5 cm.) up the forehead, was removed and the dura was exposed under this area. The patient was given a transfusion. Foreign protein therapy was instituted. Despite adequate supportive therapy, the process progressed. Symptoms of headache and vomiting suggested meningeal involvement. By December 16 there was considerable proptosis of the left eye, with chemosis. This suggested thrombosis of the cavernous sinus. Roentgen examination showed an extension of the process into the calvarium. The patient's condition became increasingly unsatisfactory and she died on Jan. 5, 1938.

Necropsy revealed osteomyelitis of the frontal, and parietal bones and of the wings of the sphenoid bone, abscess in the left temporal lobe, bilateral bronchopneumonia, cloudy swelling and fatty degeneration of the liver and cloudy swelling of the kidneys. The diploe of the frontal bone was extensively suppurative. There was no thrombosis of the cavernous sinus.

Case 2.—J. S., a white girl aged 8, was first admitted to the Research and Educational Hospital on Sept. 9, 1938. In November 1937 she had had scarlet fever. Ten days later a swelling had occurred above and behind the left eye. Treatment with hot applications had not resulted in any improvement. Incision had been made into the region of the supraorbital ridge. The wound had never healed, and a greenish purulent discharge had continued to ooze from it. Examination (by Dr. Hans Brunner) revealed a longitudinal fissure 1 cm. in length. There was a surrounding area of redness and edema on the upper eyelid and across the midline. The impression at this time was of osteomyelitis of the frontal bone secondary to frontal sinusitis.

On September 10 the patient was operated on by a supraorbital approach. The sequestrum was found fused to underlying bone, and operation was stopped on account of the poor condition of the patient. Destruction of the supraorbital arch was found, but the depth of the necrosis was not determined. There was no frontal sinus. Biopsy showed a chronic inflammatory process. The red blood cells numbered 3,850,000 and the white blood cells 12,200 per cubic millimeter. Postoperatively, the temperature went to 99.8 F. for a few days. The patient was discharged on the thirteenth day.

On Jan. 11, 1939 the patient was readmitted. The sequestrated bone was coagulated with diathermy through the spread wound, to facilitate separation. On January 12 the wound was reopened and the sequestrum inspected. The sequestrum was found to be smaller than at the previous operation. An opening was made in the processus frontalis maxillae, and the osteomyelitic area drained into the nose. The patient was discharged on the fourth postoperative day and readmitted on February 13 for a course of fever therapy to hasten separation.

On Nov. 11, after another readmission, she was again operated on. The sequestrum (just lateral to the midline) was removed. It involved only the outer table. The cavity thus produced was opened into the nose. The central portion of the wound was left open for drainage.

Fig. 2.—Roentgenogram showing operative defect after a sequestrum has been removed. Note the absence of frontal sinuses.

COMMENT

It is well recognized that osteomyelitis of the frontal bone may occur as a complication of smallpox, as a manifestation of a hematogenous extension of a generalized osteomyelitic disease or secondary to trauma, but its occurrence in the absence of a predisposing frontal sinusitis must be rare. This most probably explains its infrequency prior to pubescence, the time of life when the frontal sinus becomes well developed. In the case of the young patient herein described, the localization of the process in the outer table is most probably explained by the greater porosity of the outer table of the bone as opposed to the more vitreous character of the inner table. This case presented a special therapeutic problem,

since the usual sinal approach was not required and the infectious process was in close proximity to the superior saggital sinus. It is of interest to note that the process developed in the midline at a point away from the place of origin of the frontal sinus.

The other case doubtless represents (in retrospect) the fulminating type of osteomyelitis of Williams and Heilman.[6] Here no surgical procedure could have been adequate to stop the advance of the infection.

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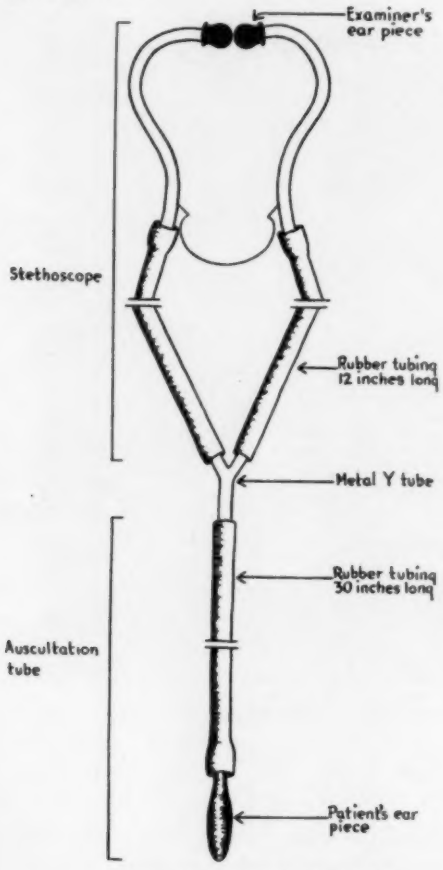
6. Williams, H. L., and Heilman, F. R.: Spreading Osteomyelitis of the Frontal Bone Secondary to Disease of the Frontal Sinus, with a Preliminary Report as to the Bacteriology and Specific Treatment, Arch. Otolaryng. **25**:196 (Feb.) 1937.

Clinical Notes; New Instruments and Technics

IMPROVISED AUSCULTATION TUBE

JOHN E. ALEXANDER, M.D., ARLINGTON, VA.

In the September issue of the ARCHIVES OF OTOLARYNGOLOGY, I noted a description of an instrument devised for holding the ear piece of the auscultation tube in place in the physician's ear.



Improvised ausculation tube.

For the past two years I have been using for this purpose an old stethoscope with a metal "Y" attached to the end of the auscultation tube. With this instrument the sounds produced by inflation of the eustachian tube are audible in both ears of the examining physician, and both hands are free, just as described in the previously mentioned clinical note.

Arlington Medical Center.

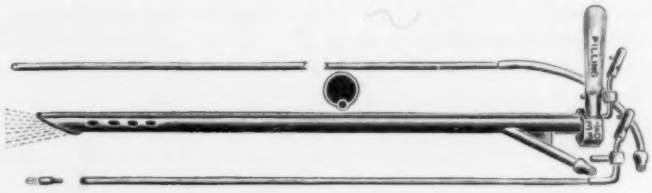
When part of the stethoscope is used for assembling a satisfactory auscultation tube, one instrument is utilized for two distinct purposes, and the change from one to the other can be made in a few seconds. Every physician has a stethoscope, and I cannot see the need for additional equipment when this arrangement is perfectly satisfactory.

NEW INFANT BRONCHOSCOPE AND ESOPHAGOSCOPE

PAUL HOLINGER, M.D., CHICAGO

The safety of bronchoscopic or esophagoscopic examination of newborn infants depends on the employment of small caliber instruments. The use of a new miniature lamp and lamp carrier has made possible a significant decrease in the external diameter of the infant bronchoscope and esophagoscope herein described, without decreasing the working lumens.

The lamp, 1.7 mm. in diameter and 15 mm. in over all length, is detachable from its carrier. The carrier and the lamp are of the same diameter and rest in a grooved channel, whose sides are carried into the lumen of the bronchoscope just far enough to hold them in place throughout the length of the tube.



Infant bronchoscope, aspirator, light and light carrier.

The bronchoscope is of the Chevalier Jackson type,[1] 3.5 mm. in internal diameter, 5.5 mm. in external diameter and 25 cm. or 30 cm. in length. The unusual smoothness of the distal tip of the instrument permits easy introduction of the tube between the vocal cords, as there is practically no increase in the diameter of the tube at this point. The light carrier canal and the smooth construction of the distal tip are patterned after the Negus "suckling bronchoscope."[2] The proximal handle is reduced in size to give the instrument proper balance.

The esophagoscope is of the Chevalier Jackson "full lumen" type, with the small light and light carrier previously described. Its lumen is 3 by 4 mm. and its length 30 cm. The aspirating canal has the usual Tucker thumb valve.

Because of the small size of the light and light carrier, a significant decrease in the size of the outer dimension of both instruments is possible. This feature is especially important in the bronchoscope, as it facilitates its introduction through

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Presented at the meeting of the American Bronchoscopic Society May 27, 1939.

From the bronchoscopic clinics of the Children's Memorial Hospital, St. Luke's Hospital and the Research and Educational Hospital of the University of Illinois.

1. Jackson, C.: Bronchoscopy and Esophagoscopy, Philadelphia, W. B. Saunders Company, 1927.

2. Thomson, St. C., and Negus, V. E.: Diseases of the Nose and Throat, ed. 4, New York, D. Appleton-Century Company, Inc., 1937.

the larynx of the newborn infant and lessens the possibility of trauma without sacrificing visibility or the efficiency of the instrument.

The George P. Pilling & Son Company, of Philadelphia, who constructed this infant bronchoscope and esophagoscope contributed valuable suggestions.

A METHOD OF LABYRINTHECTOMY

ERIC GUTTERIDGE, M.D., F.R.C.S. (EDIN.), F.R.A.C.S., D.L.O. (LOND.)
MELBOURNE, AUSTRALIA

Labyrinthotomy, or drainage of the labyrinth, would describe more accurately the various operations on the labyrinth. The presence of the facial nerve superimposed on the labyrinthine capsule precludes a complete exenteration of the membranous labyrinth unless the Uffenorde method is adopted. This isolates and exposes the facial nerve, lifts it from its bony bed and places it aside, where it will not be subject to injury during operation. After the semicircular canals and the vestibule have been opened and the membranous labyrinth curetted away, the facial nerve is replaced on its bony viaduct. Uffenorde has never encountered a lasting facial paralysis as a result of operation by this method.[1]

All other classic operations on the labyrinth strictly preserve the integrity of the bony canal protecting the facial nerve.

There are two major problems of localization involved in an attack on the labyrinth:

1. The position and extent of the vestibule and its relation to the visible landmarks—the lateral, or external, semicircular canal and the skeletonized facial ridge. Kirschner[2] stated that some indication of the position of the vestibule may be gleaned from the relation of the lateral semicircular canal to the facial ridge. If the canal is exactly horizontal, the vestibule will be found directly above the facial ridge. If it inclines forward, the vestibule will be found more beneath the facial nerve, and the tip of the cochlea is accordingly lower. If, on the contrary, it inclines backward, the vestibule will be turned backward and the point of the cochlea directed upward. Spalteholz' "Hand Atlas of Human Anatomy" shows the facial nerve crossing the equator of the vestibule with a small upward obliquity.[3]

As the small oval cavity of the vestibule is from 3 to 5 mm. in size, it is obvious that only an approximate position can be mapped out.

2. The second problem is to discover at what depth the vestibule is placed beneath the canal of the facial nerve. With the facial ridge lowered to the safety limit, what depth can be allowed for the integrity of the bony viaduct carrying the facial nerve? A too superficial attempt to enter the vestibule will endanger the facial nerve; a too deep excavation of bone will pass medially to the vestibule, enter the internal auditory meatus and possibly injure the facial nerve as it passes through the meatus to gain entrance to the facial canal in that vicinity.

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1. Kirschner, M.; Lautenschläger, A., and Kleinschmidt, O.: Operative Surgery: The Ear, Air Passages and Neck, translated by I. S. Ravdin and G. M. Coates, Philadelphia, J. B. Lippincott Company, 1937, vol. 3, p. 71.

2. Kirschner, Lautenschläger and Kleinschmidt,[1] p. 65.

3. Spalteholz, W.: Hand Atlas of Human Anatomy, translated by L. F. Barker, ed. 7, Philadelphia, J. B. Lippincott Company, 1933, vol. 3, fig. 961.

The classic types of operation seek the vestibule from two aspects:

1. The Jansen-Neumann, Richards and Beck technics expose the semicircular canals, open their lumens and follow the canals to the vestibule.

The Jansen-Neumann operation, entering Trautmann's area and pressing backward the dura of the cerebellar fossa, shaves down the bone of the posterior surface of the pyramidal petrous bone until the canals leading to the vestibule are opened. A difficulty arises when the lateral sinus is placed abnormally forward, limiting the space available for chiseling the bone medially and forward. A curved or oblique chisel may be required to remove the bone beneath the facial viaduct. The superior petrosal sinus is endangered if the excavation is too high and is accompanied with troublesome hemorrhage. The internal auditory meatus, with the facial nerve, is medially adjacent to the line of attack and may be opened accidentally. The mastoid emissary vein entering the lateral sinus is in the field and may cause hemorrhage, obscuring the operative area. However, the main difficulty lies in not knowing the exact point at which to enter the chisel.

The Richards and Beck operations dissect the three canals from the surrounding petrous bone and then enter the exposed canals to find the vestibule.

2. The Hinsberg operation commences in front of the facial ridge by opening the cochlea and vestibule in the region of the promontory. The fenestra vestibuli and fenestra cochleae are joined by chiseling the small plate of bone separating them. A probe passes into the opening of the lateral semicircular canal. The lateral wall of this canal is then carefully removed until the lumen appears. There is a risk of fracturing the lateral canal and, with it, the facial canal in immediate caudal juxtaposition. The ampullae of the lateral and superior semicircular canals are both exposed and opened. The vestibule is not further opened, and it is questionable whether a sufficiently adequate drainage of the labyrinth is obtained by this method.

In 3 labyrinthectomies of a recent series of 7 performed by the Jansen-Neumann method, difficulty was experienced from hemorrhage from the mastoid emissary vein, from the superior petrosal sinus and from a lateral sinus situated so far forward as to restrict the operative field—Trautmann's area. A considerable time was taken in searching for the tiny openings of the superior and posterior canals.

A dissection of the labyrinth discloses that a bent probe entering an enlarged fenestra vestibuli (oval window) will pass into the vestibule and will delimit the position, size and depth of the cavity. It will also demonstrate the position of the vestibule relative to the facial ridge and the safe limits of the exenteration of bone to be performed. With the probe in situ, the bone can be chiseled behind the facial ridge, inward and forward, with relative absence of danger to the facial nerve and perfect orientation. It is only necessary to cut down on the tip of the bent probe, and the posterior limb of the lateral semicircular canal appears as it opens into the vestibule. There is no necessity to expose the cerebellar dura or the lateral sinus.

Figure 1 *A* is a representation of the vestibule, crossed by the viaduct of the facial nerve, with the lateral and superior semicircular canals superimposed. This represents the normal position of the vestibule in its relations to the facial nerve. The fenestra ovalis and fenestra cochleae are not indicated.

The operation proceeds as does a complete radical mastoidectomy. The facial ridge is shaved down to the permissible limit. The floor of the external auditory meatus is lowered.

The stapes is extracted.

The oval and the round window are exposed. The small bridge of bone separating them is broken through with a small chisel and removed with a curet to expose the lower extension of the vestibule. Any overhanging edges are removed.

A fine probe, bent at a right angle 6 mm. from the tip, is now inserted into the vestibule and passed beneath the viaduct of the facial ridge into the depths of the vestibule proper. The dimensions and boundaries of the vestibule can now be mapped out with accuracy. The depth of the facial ridge medially is measured. The direction of the vestibule from the oval window is evident (fig. 1 *B*).

With these data and with the probe in place as a guide, no errors in orientation are possible. It remains only to excavate toward the tip of the probe guide to open the vestibule.

The bone adjacent to the posterior extremity of the lateral semicircular canal is removed with a gouge or a power-driven burr, directly toward the tip of the

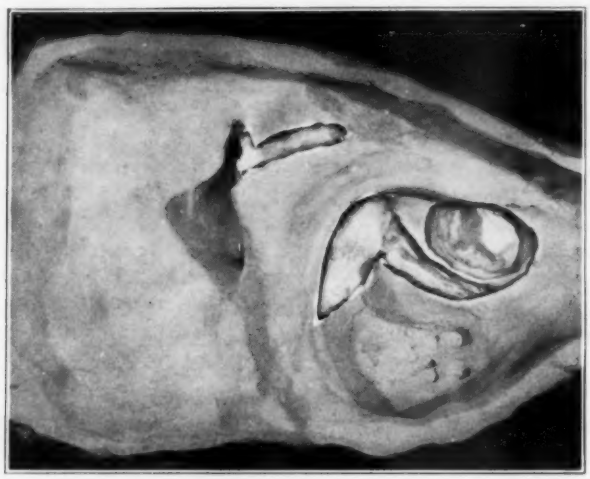

Fig. 2.—The operative field after the labyrinthectomy has been completed. The vestibule, the lateral semicircular canal and the cochlea have been opened.

probe guide. The direction will be inward and forward beneath the facial ridge, the inner limit of which is known from the position of the guide. The posterior limb of the lateral semicircular canal first appears, as it opens into the vestibule. The tip of the probe appears in this opening as it is enlarged. The vestibule is widely opened with the curet.

The lateral semicircular canal may be followed and opened by chiseling away the upper rim. If it is thought desirable, the cochlea may be drained by removing the promontory.

This completes the operation. A wide entrance has been made into the vestibule in front of and behind the facial canal. Adequate drainage of the semicircular canals has been obtained.

Figure 2 shows the operative field after the labyrinthectomy has been completed. It also demonstrates that a small amount of bone has been removed and that the posterior surface of the temporal pyramid, with the lateral sinus and the cerebellar dura, has not been exposed during the operation.

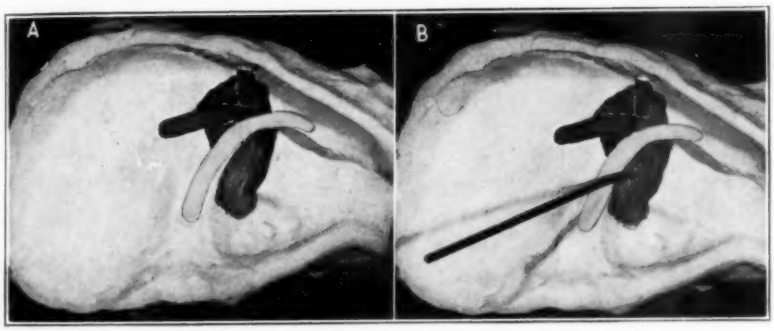

Fig. 1.—*A*, a representation of the vestibule and its relations to the facial nerve and the lateral and the superior semicircular canal; *B*, the probe guide in the vestibule. The oval and the round window have been joined.

The operative field is swabbed with a solution of trinitrophenol and acetone, and all fragments of mucosa removed from the remaining labyrinthine wall of the middle ear. A paste consisting of 1 part of bismuth subnitrate, 2 parts of iodoform and enough paraffin to make a soft paste is smeared over the field, and the cavity is packed with ribbon gauze soaked in an emulsion of acriflavine hydrochloride in liquid petrolatum (1 in 1,000). The wound is then sutured. The gauze is removed in small portions daily, so that it is all withdrawn by the seventh day. It is then replaced daily.

This method enables a labyrinthine drainage to be rapidly, efficiently and safely performed. There is a minimum of disturbance to the patient.

SUMMARY

A labyrinthotomy, which commences by opening of the vestibule anterior to the facial ridge and in which a bent probe is passed beneath the facial canal as a guide to the location of the vestibule, is described.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

CHRONIC PROGRESSIVE DEAFNESS, INCLUDING OTOSCLEROSIS AND DISEASES OF THE INNER EAR

GEORGE E. SHAMBAUGH JR., M.D.
CHICAGO

A growing interest in the surgical treatment of otosclerosis is evident in the publication in 1939 of a larger number of articles dealing with this subject. The increased interest in Ménière's syndrome noted the previous year continued during 1939. The relation of vitamins to deafness received enough attention in 1939 to warrant a special section in this review.

As in previous summaries, I have directed my attention to articles which contribute something new to the knowledge of progressive deafness and in editorial comments, in parentheses, I have given my evaluation of many of the contributions. The material is reviewed in the following order:

Otosclerosis
- Pathology and Etiology
- Treatment

Labyrinthine Deafness
- Pathology
- Hereditary Nerve Deafness
- Traumatic Nerve Deafness
- Nerve Deafness from Infections
- Nerve Deafness from Avitaminosis
- Nerve Deafness from Other Causes
- Treatment of Nerve Deafness
- Ménière's Syndrome

OTOSCLEROSIS

Pathology and Etiology.—Wittmaack[1] contributes a valuable discussion of the histology of otosclerosis, which is of sufficient importance to warrant the following detailed abstract.

1. Wittmaack, K.: Considerations About the Otosclerosis Problem, Hals-, Nasen- u. Ohrenarzt (pt. 1) **30**:337 (Oct.) 1939.

Wittmaack takes exception to the statement made by Nager and M. Mayer that since otosclerosis is identical with osteodystrophy (Paget's disease) there is no longer a problem of otosclerosis. One important difference between the two diseases, Wittmaack points out, is that different stages of osteodystrophy can be found in the same specimen and sometimes even in one section from this specimen whereas only one or two stages of otosclerosis can be recognized in the same specimen. An exception occurs if a fresh otosclerotic focus develops next to a subsiding or subsided focus.

Wittmaack points out that the interpretation of the histologic picture depends on the cellular elements and the circulatory system and not on the intercellular substances (osteoid and collagen), which are dependent on the cells and circulation and not vice versa. Thus, otosclerotic foci with rich vascularization and large accumulations of cells are progressing rapidly, while diminishing vascularity and fewer cells point to a subsiding process, which with the formation of new bone may even reach a quiescent stage.

Wittmaack presents sections from an otosclerotic focus of an 8 year old girl who died of diphtheria and whose temporal bones showed the triad characteristic of the most active stage of otosclerosis:

1. One or more greatly enlarged veins entering the focus, with areas of bone resorption around the branches of the veins that have a varicose appearance.

2. Unusually large and numerous bone cell cavities with very striking large bone cells ("sievelike" perforation of the bone).

3. Extensive blue staining of the bone substance surrounding the vessels and areas of resorption.

All three findings must be present to substantiate a diagnosis of otosclerosis. A single enlarged vein or a small blue bony edge with a few enlarged bone cell cavities is not sufficient to warrant a diagnosis of otosclerosis (contrary to certain other authors).

The question is, does the dilatation of the veins result from or does it cause the otosclerotic bone change? Wittmaack believes that the bone changes are secondary to the dilatation of the veins, for the following reasons:

1. There may be relatively large dilated veins with small foci of resorption, suggesting that the latter are secondary rather than primary.

2. The enlarged vessels are apparently veins and not arteries. In the early stages the artery is so compressed by the dilated vein as to be invisible. Such enlarged veins entering or leaving a focus have never been described in osteodystrophy. Moreover, in the early stage of osteodystrophy, the connective tissue has relatively few cells, without

enlarged vessels but with typical Howship's lacunae. These lacunae are never formed in the early otosclerotic focus.

In one specimen Wittmaack found a picture not previously described: At the typical place for an otosclerotic focus there was the enlargement of the veins always found in the disease. Around these veins were areas of resorption, with slightly red staining of the bone around the resorbed areas. The areas of resorption consisted of fibrous tissue containing many crowded large cells of the same appearance as the surrounding bone cells. Wittmaack interprets these conditions as the earliest stage of otosclerosis. The slowing down of the blood stream and increased pressure in the dilated veins resulted in local acidosis and halisteresis (decalcification), with formation of areas of bone resorption. The red staining of the surrounding bone is characteristic of halisteretic bone softening. If the process is completed and local alkalosis supervenes, a sediment of lime is deposited in the softened bone, taking a more or less intense blue stain. From this point on the process has an undulating course, with periods of acidosis alternating with periods of alkalosis, but since the bone now stains deeply blue, there is only a reduction in the intensity of the blue staining, and a reversion to red staining cannot occur after the initial stage. Wittmaack has found red stained bone only twice in a large number of otosclerotic specimens.

Sometimes, if much lime has been deposited, foci of resorption due to the formation of Howship's lacunas are found on the borders of the focus. This occurs in older foci particularly when the vein no longer contains blood, and is apparently the result of a secondary nutritional disturbance of the bone due to the abnormal deposit of calcium or to the circulatory disturbance.

Wittmaack speaks of reversed blood flow rather than of blood stasis, as he did twenty years ago, for true blood stasis cannot last any length of time without thrombosis or reversal of the flow. On the time and intensity of the reversal depend the extension and intensity of the otosclerotic process. Small otosclerotic foci that have subsided quickly are found with remarkable frequency. They are easily explained: The reversal of the blood stream was brief, and anastomoses formed quickly.

Wittmaack deals with objections that have been raised to his theory. Some say that red-stained bone is new, not softened, bone. Wittmaack points out that red staining is due to a failure of the stain to combine with the calcium of the bone and that one cannot determine by staining whether bone is old or new but must rely on the appearance of the bone cells. In new bone the cells are arranged in a row along the red-stained bony margin, for the bone is being formed by the osteoblasts. In bone resorption, on the other hand, the cells leave the cell cavities and accu-

mulate irregularly between the vessel and the dissolving bone edge. The change from dissolution to new bone formation can take place relatively quickly.

Otosclerotic foci in the capsule of the cochlea are especially suitable for comparison with osteodystrophic foci, which also occur in this region. The differences between the two lesions are: First, otosclerotic foci are usually situated in the enchondral layer, whereas the osteodystrophic process develops regularly at first in the periosteal layer. Second, the otosclerotic focus always develops around vessel canals and radiates out along the branches of the vessels, whereas osteodystrophy develops from multiple small dispersed foci by typical Howship's lacunae, with the result that the bone of the labyrinthine capsule is interspersed with many large marrow spaces, such as are never found in otosclerosis. Finally, the osteodystrophic process is diffuse, whereas the otosclerotic process is sharply separated from entirley normal areas. The fact that otosclerotic and osteodystrophic bone have been observed in the same specimen must be regarded as due to pure coincidence and to the fact that otosclerosis is a common disease.

[Wittmaack's article is beautifully illustrated throughout and should be read in the original, with the histologic photographs to supplement the text. The weak point in Wittmaack's theory of the genesis of otosclerosis is that he does not satisfactorily explain the reversed venous flow to which he attributes the bone changes.]

Nager [2] reiterates his views on otosclerosis. On the basis of histories of 1,146 cases of otosclerosis supplemented by many sections of temporal bones he states that otosclerosis occurs in around 10 per cent of all adults but that in only a small proportion of these is the disease manifest. In the great proportion otosclerosis remains latent, without symptoms, the foci being unsuspected until the temporal bone is sectioned. The sex ratio is 1 male to 1.8 females. The onset of symptoms in over half the cases occurred between the ages of 16 and 30. Hereditary and familial deafness were found in over 50 per cent. In 10 per cent the deafness remained confined to one ear for many years; this proportion was confirmed by histologic study. A definite increase in deafness from pregnancy was observed in only 80 of 383 married women with otosclerosis.

Although the ultimate cause of the disease is not yet known, Nager regards the genesis as clear: Otosclerosis represents osteodystrophy confined to the labyrinthine capsule, progressing with extreme slowness and showing all the signs of pathologic resorption and deposition of bone, with replacement by more or less mature bone. When the process

2. Nager, F. R.: Clinical Manifestations and Pathologic Anatomy of Otosclerosis, Nord. med. (Hygiea) **2**:1703 (June 3) 1939.

traverses the annular ligament (or extends into the nerve canals), an impairment of hearing occurs.

Sparer [3] presents the hypothesis that otosclerosis is a local bone disease due to local alterations in circulation as a result of vasomotor imbalance of the nerves controlling the blood vessels, these nerves in the tympanic plexus being connected with the sphenopalatine ganglion via the greater superficial petrosal nerve. For the removal of abnormal impulses from the sphenopalatine ganglion, he advises thorough eradication of any nasal disease. Injection of alcohol into the ganglion may also be employed to disrupt efferent irritating impulses. A small series of patients are being treated along these lines, Sparer says, with encouraging results.

[This new and different theory of the cause of otosclerosis thus far entirely lacks pathologic, clinical and experimental proof.]

Bunch [4] followed a small group of patients with clinical otosclerosis by means of repeated audiometric tests for periods up to six years. In some there was no measurable loss of hearing. In the few in whom deafness increased, no obvious factor was noted which might account for the increase. The 2 who showed definite improvement in hearing underwent serious surgical operations. Of particular interest were 6 women who were tested before and after pregnancy. In no case was there a measurable increase in the hearing defect as a result of the pregnancy.

[My experience coincides with that of Bunch; i. e., in otosclerosis the hearing may decline rapidly for a year or two and then remain stationary for years. Presumably the otosclerotic focus goes through a corresponding period of active enlargement followed by a period of quiescence.]

Frenkel [5] cites as evidence of the hereditary nature of otosclerosis the case of a woman of 56 years with unilateral involvement. Otosclerosis occurred in a maternal aunt, in a maternal uncle and in two sisters who are twins. One of these twins is married and has a son with otosclerosis.

Schwarz [6] found a greater frequency of otosclerosis in persons of the asthenic type than in persons of the pyknic type. He regards the disease as due to an inferiority of the mesenchyme.

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3. Sparer, W.: The Pathogenesis of Otosclerosis, Laryngoscope **49**:1199 (Dec.) 1939.

4. Bunch, C. C.: The Progress of Deafness in Otosclerosis, Laryngoscope **49**:793 (Sept.) 1939.

5. Frenkel, I. S.: Etiology of Otosclerosis, Vestnik otol., 1938, no. 5.

6. Schwarz, M.: Otosclerosis and Body Form, Hals-, Nasen- u. Ohrenarzt (pt. 1) **30**:147 (May) 1939.

Engstrom [7] found otosclerosis in 4 of 34 consecutive autopsies on persons between the ages of 30 and 70. In 2 of the 4 cases the subject had been hard of hearing, apparently as a result of thickening of the annular ligament from otosclerotic changes around the oval window from a focus in that region, although ankylosis had not yet occurred. In 2 hearing had been normal, and the otosclerotic changes were diffused throughout the otic capsule in the form described by Manasse as "blue strands." The author's findings are in agreement with those of others who find otosclerosis frequently in routine autopsies (Fowler Jr., 5 per cent; M. Weber, 10 per cent and Nager, 10 per cent).

Frenckner [8] took motion pictures of the drum membrane through the Siegle-Brüning speculum and found that in 2 normal persons the umbo moved a maximum distance of 15 to 17 mm. with changes in air pressure whereas in a patient with otosclerosis the umbo moved only 4 mm. He does not believe that the mobility of the malleus could be impaired to this extent by fixation of the stapes alone and suggests that there were otosclerotic changes in or near the incudomalleolar joint.

Greifenstein [9] collected statistics from a number of clinics and found that pregnancy had been interrupted because of otosclerosis in 79 cases. All other otolaryngologic indications for interruption of pregnancy, including tuberculosis of the larynx, were less frequent than otosclerosis. Concerning the individual cases in which pregnancy was interrupted, no details are given.

[The importance of pregnancy in increasing deafness in otosclerosis has probably been overemphasized. Thus, Bunch found no increase in the deafness of 6 women and Nager found an increase from pregnancy in only 1 of 4.8 women, while in my own experience pregnancy has resulted in a definite increase in the deafness in only about 1 patient in 5.]

Treatment.—Medical treatment of otosclerosis was recommended by several authors in 1939.

Mortimer, Wright, Thomson and Collip,[10] having noted that atrophic rhinitis and constitutional deafness sometimes occur in the same patient and having found that estrogens are of value in atrophic rhinitis, treated a series of 39 females and 16 males for constitutional deafness by daily insufflation of 1 cc. of oil containing 1,000 international units of an

7. Engström, H.: On the Frequency of Otosclerosis, Acta oto-laryng. **27:** 608, 1939.

8. Frenckner, P.: Movements of the Tympanic Membrane and of the Malleus in Normal Cases and in Cases of Otosclerosis, Acta oto-laryng. **27:**587, 1939.

9. Greifenstein: Interruption of Pregnancy and Sterilization for Reasons of Health in Otosclerosis, Hals-, Nasen- u. Ohrenarzt (pt. 2) **49:**42 (July) 1939.

10. Mortimer, H.; Wright, R. P.; Thomson, D. L., and Collip, B.: Canad. M. A. J. **40:**17 (Jan.) 1939.

estrogen for periods of three to six months. They treated some of the patients intermittently for two years. A control group of 8 males and 6 females were observed during the same period. The authors did not attempt to differentiate the types of deafness for which treatment was given except to state that their cases were "run of the mine" clinic cases and were mostly diagnosed as either "otosclerosis" or "nerve deafness." In age the patients ranged from the second to the sixth decade. Before and during treatment, at intervals of one to two months, the hearing was tested with an audiometer in a room adjacent to the clinic that was "by no means a quiet room but quieter than the main clinic rooms." The results of the treatment are summarized by giving the difference in decibels for each frequency in each ear between the first and the last audiogram. The authors conclude:

> Statistical examination shows that when all the data for 512, 1024, 2048 and 4096 cycles are considered, the treated males do, on the average, improve slightly in hearing while the untreated males slightly deteriorate. At these same frequencies . . . the treated females do on the average improve slightly in hearing. The untreated females appear to deteriorate. . . . This would seem to indicate that . . . sex hormones may still play a rôle in the physiology of hearing . . . at least when constitutional defective function exists.

[Because of the widespread attention, including newspaper publicity, which this article attracted and because in a small series of cases of otosclerosis I was unable to demonstrate any effect on the hearing from the use of estrogens as advised, the results obtained by the authors were closely scrutinized. In 9 of 16 treated males the average change for the frequencies 512, 1024, 2048 and 4096 was less than 10 decibels; in other words, the change was within the limits of normal variation. Of the 7 treated males who showed a change of more than 10 decibels for these frequencies, 3 showed losses of —11, —16 and —20 decibels, while 4 showed a gain of +16 decibels. Of the 8 males in the control group, 4 showed a change of less than 10 decibels, 1 a gain of +16 decibels and 3 losses of —11, —19 and —22 decibels. While it is true that "statistically the treated males, as a whole, showed a slight improvement and the untreated males a slight deterioration," when one considers the individual cases one is extremely doubtful that the treatment can be regarded as of proved value. The published results for the treated and untreated females show essentially the same changes as those for the males.]

Guggenheim [11] states that over 50 per cent of women with otosclerosis lose markedly in hearing during or after pregnancy. This, he believes, is due to an insufficient intake of calcium during pregnancy,

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11. Guggenheim, L. K.: On the Diagnosis and Treatment of Otosclerosis, Am. J. Surg. **43**:156 (Oct.) 1938.

resulting in decalcification of the otic capsule around the otosclerotic focus, with spread of the focus. The same explanation is offered for the frequent onset of otosclerotic deafness at puberty, when growth is rapid and the intake of calcium may be inadequate. The author suggests that increase in an otosclerotic focus may be arrested by correction of the diet to normal requirements with the addition of: vitamin C, 1,000 to 3,000 units daily; calcium, 3 to 6 Gm., and phosphorus, 6 to 12 Gm., daily, in the form of dicalcium phosphate ($CaHPO_4$); vitamin D, 3,000 to 6,000 U. S. P. units daily, and when tinnitus is present, 1,000 to 2,000 Sherman-Chase units of vitamin B_1 daily. The author states that there is no thought of improving hearing and that it will require many years' work with a large series of patients to prove that the loss of hearing can be arrested.

[The author's statement of the frequency of increased deafness from pregnancy in otosclerosis does not agree with the statistics of others (see comment, p. 441). The value of the dietary treatment of otosclerosis is, of course, purely hypothetic, since no one has yet published any experimental, pathologic or clinical proof.]

Screlercnikov believes that diet and the endocrine and the vegetative nervous system play important parts in the etiology of otosclerosis. He advises the use of endocrine substances, vitamins and lysates in the prophylaxis and treatment of otosclerosis.

Davis and Rommel [13] treated 28 patients who had acute blocking of the eustachian tube, associated with tinnitus and usually with marked deafness, by catheterization of the eustachian tube and massage of the drum membrane and with injections of a solution of prostigmine methylsulfate. Usually the condition cleared up with not more than five injections of the drug.

In 29 cases of chronic deafness (diagnosis not indicated) the usual inflation and massage, with treatment of any associated sinusitis, were combined with injections of a solution of prostigmine methylsulfate, with improvement in hearing or tinnitus in all cases. The injections should be given two or three times a week for both acute and chronic conditions. Acute conditions are cured in five treatments or less, while chronic conditions require that the treatment be continued indefinitely.

[This article also received widespread attention, including newspaper publicity. However, since deafness due to blocking of the eustachian tube generally clears up with inflations alone, the value of

12. Footnote deleted.

13. Davis, T. C., and Rommel, J. C.: Treatment of Deafness and Contiguous Nervous Disorders with Prostigmine, Arch. Otolaryng. **29**:751 (May) 1939.

adding injections of the prostigmine salt to the treatment of acute deafness is not proved. No treatment for chronic deafness can be accepted as of proved value without audiometric control. Unless the authors can report audiometric improvements greater than the normal variations, their treatment cannot be regarded as of proved value. In my own hands the use of prostigmine has not thus far been of demonstrable value either for loss of hearing or tinnitus in any case.]

Articles on the surgical treatment for otosclerosis may be grouped into those dealing with some form of fenestration of the labyrinth and those in which other methods for improving the hearing are described. In the latter group is a contribution by M. Meyer,[14] who believes that the auditory improvements on opening the labyrinth described by Holmgren and Sourdille are due to decompression of the intralabyrinthine fluids. He accordingly attempts to decompress these fluids by removing 30 to 50 cc. of spinal fluid by suboccipital puncture. Six patients treated in this manner showed remarkable improvement in hearing. Four of the 6 had otosclerosis; in the other 2 the cause of the deafness was not clear. The hearing gain lasted from a few weeks to several months, the longest period being six months. In all, 20 persons with otosclerosis were treated, and in only 2 did the treatment fail.

[Without audiometric substantiation it is difficult to know what the author means by "remarkable improvement" and it is impossible to evaluate his results. There is no histologic, experimental or clinical evidence of increased pressure of the spinal and intralabyrinthine fluids in otosclerosis. Why does the author use suboccipital puncture instead of the safer and easier lumbar puncture?]

Alonzo and Chiarino[15] report a total of 19 cases of otosclerosis in which parathyroidectomy resulted in an improvement in hearing followed by a deterioration, although generally the hearing remained above the preoperative level. The improvement was mainly in hearing for voice in young people.

[The same authors previously reported a smaller series in which treatment was by this method. Again, without audiometric substantiation one cannot accept the statement of hearing improvements.]

When there is evidence of hearing in a malformed ear, Padgett[16] suggests the surgical construction of an external auditory canal to

14. Meyer, M.: A New View About Deafness in Otosclerosis and a New Treatment, Acta oto-laryng. **27**:1, 1939.

15. Alonzo, J. M., and Chiarino, A.: Otosclerosis and Otospongiosis: Influence of Parathyroidectomy on the Hearing, Acta oto-laryng. **27**:123, 1939.

16. Padgett, E. C.: An Operation for Possible Alleviation of Certain Cases of Congenital Deafness and Certain Types of Acquired Chronic Deafness, Ann. Otol., Rhin. & Laryng. **48**:113 (March) 1939.

allow sound vibrations to reach the middle ear. A skin flap, free below and attached above, is made from the hairless skin over the mastoid. A canal is made through the mastoid process into the antrum, and the skin flap is rolled around a tube and inserted into the aditus. At a later date the postauricular opening may be transferred to a position in front of the auricle, the normal position of the meatus. The author mentions that this same operation has been used by O. J. Dixon in certain cases of chronic deafness.

[When there is congenital atresia, or absence, of the external auditory canal, the drug membrane and ossicles are generally absent or malformed, and construction of a meatus cannot be expected to restore normal hearing. If both ears are malformed or if the malformed ear is the better hearing ear, any slight improvement in hearing is desirable and an operation such as that described would be justified.]

Diamant [17] observed a patient with otosclerosis and a central perforation of the drum membrane who showed an undoubted increase in deafness after the application of a cellophane prosthesis, with an improvement in hearing when the prosthesis was removed. He therefore performed a paracentesis of the drum in a patient with otosclerosis and secured subjective and objective improvement in hearing. The treatment has been used for 10 patients. To reduce the intralabyrinthine pressure, the author filled the middle ear with boric acid (powder), which had a further improving effect on the hearing. Ten per cent cocaine and glycerin were also used to reduce the pressure, but the results were doubtful.

[Paracentesis of the drum membrane has been tried before in otosclerosis but has been given up as valueless.]

Hughson [18] discusses the surgical treatment of deafness and expresses the belief that the results thus far attained justify the attempt to relieve deafness surgically in patients who are suitable for operation. The two surgical procedures discussed are fenestration and blocking of the round window niche. Of the latter procedure the author says:

> In a small series, 35 cases to date, there have been no infections, no patient has suffered further impairment, all frequencies have been affected and the maximum useful improvement obtained is as great as that reported for fistulization in any case. The effect is slow in developing, but when once begun it has continued to the limit of time any case has been observed, two years.

[The author's claim of hearing improvements as great as those after the fenestration operation has not been borne out by any of his published case reports.]

17. Diamant, M.: Concerning a Hearing Improving Treatment in Otosclerosis, Nord. med. (Hygiea) **1**:321 (Feb. 4) 1939.

18. Hughson, W.: Surgical Treatment of Deafness, Arch. Otolaryng. **30**: 497 (Oct.) 1939.

Holmgren [19] contributes several articles on the surgical treatment of otosclerosis, in which he describes new clinical and experimental work. Influenced by his previous failures to secure permanent results, he has used various methods for keeping the fistula open. One method consisted of a radical mastoid operation in the first stage with or without a Thiersch graft. After the cavity had epidermized, a fistula was made and covered with the epidermal lining of the cavity. A second method consisted of an exenteration of the mastoid cells, the wound being filled with paraffin (melting point 42 C.). In the second stage the paraffin was removed, a fistula made and the thin cicatricial membrane which had formed around the paraffin was used to cover the fistula. Experiments were carried out on cats and monkeys, and the fistulas were covered with fat, gold leaf, paraffin or peritoneum, were left alone for some time and were then examined by Nager histologically.

It is known that in many cases fistulas caused by cholesteatoma remain open as long as the cholesteatoma is not touched and even if the cholesteatoma has been removed. This is because the fistulas have been caused by long-continued pressure on the bone. Accordingly, Holmgren is making grooves in the horizontal canals of monkeys and filling them with gutta-percha. The results are not yet known. Finally, he is using radium in the fistulas, a procedure which is giving promising results in monkeys.

In the discussion of this paper, Sourdille states that the making of a fistula is not sufficient to preserve hearing but that a labyrinthopexy as described by him must be done.

[Holmgren deserves great credit for his persistence in searching for a method of keeping fistulas open. It is clear from his writings that he has not yet found the answer; bony closure with loss of the hearing gain continues to occur with regularity.]

Nager [20] examined histologically the temporal bones of monkeys in whose semicircular canals Holmgren had made fistulas. There was generally a great tendency toward the formation of new bone, which originated nearly always in the periosteal bone. In only 2 cases were there evidences of old labyrinthitis. The operation seems not to be followed by severe suppurations and seldom by destruction of the labyrinth. The use of radium seems to reduce but not to inhibit completely the formation of new bone, and the inner ear is not injured.

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19. Holmgren, G.: The Present Standpoint of Otosclerosis Surgery, Nord. med. (Hygiea) **2**:1704 (June 3) 1939; Surgery of Otosclerosis, Acta oto-laryng. **27**:338, 1939.

20. Nager, F. R.: Demonstration Regarding Holmgren's Otosclerosis Operation in Monkeys, Acta oto-laryng. **27**:350, 1939.

Covell [21] describes experiments on enchondral bone from the otic capsule that have an important bearing on the artificial fistula of the labyrinth. Small fragments of bone from the margin of the oval window of a rabbit were transferred to the anterior chambers of the eyes of other rabbits, and the changes were observed at intervals up to seven months. After thirty-three days vascular and fibrous tissue connections to the transplant were well established, with evidence of formation of new bone and necrosis of the old bone. The new bone was all next to the periosteal bone, with none next to the enchondral bone. After ninety-nine days a thin layer of new bone lay on the free surface of the periosteal bone, but there was none on the enchondral bone. After one hundred and sixty-six days the enchondral bone, which stained poorly in the earlier specimens, again stained deeply but there was no new bone next to it.

The author believes that enchondral bone must lack the necessary chemical combinations of calcium or phosphorus to stimulate ossification, since periosteal bone, regardless of its viability, has a favorable influence on ossification. He recounts the peculiarities of the enchondral part of the otic capsule, as summarized by Nager:

> (1) With the exception of the ossicles of the middle ear, this type of bone occurs in no other locality of the adult skeleton. (2) It is to be regarded as an arrested stage in the normal development of bone in cartilage. (3) It is completely formed in the newborn and its appearance is not materially changed throughout life. (4) It is poorly vascularized bone and its metabolism must be of an extremely low grade. (5) It does not participate in the usual processes of resorption, deposition and healing of bone.

Canfield [22] cites the experiments performed by Holmgren, who covered the labyrinthine fistulas in monkeys with Thiersch grafts. This procedure was followed by prompt regeneration of bone and closure of the fistulas. Autogenous peritoneal grafts were then used, some with the peritoneal surface and some with the fibrous surface against the bone, and the fistulas closed. Mesentery with epithelium on both sides was used with the same results. Pieces of rubber placed against the bone failed to prevent regeneration. The placing of various metals against the bone has so far not given conclusive results. Finally, a small capsule of stainless steel enclosing a minute amount of radium was placed against the fistula for sixteen days, and after two more weeks the labyrinth was sectioned; although the fistula was filled with fibrous tissue, there was no evidence of bone regeneration.

The author has seen Holmgren and Lempert operate and has noted that the method used in making the fistula differs, Holmgren using a

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21. Covell, E. P.: Experimental Studies on Enchondral Bone of the Otic Capsule, Ann. Otol., Rhin. & Laryng. **48**:934 (Dec.) 1939.

22. Canfield, N.: Labyrinthine Fistulas, Arch. Otolaryng. **30**:50 (July) 1939.

scraping gouge and Lempert a dental polishing burr. He studied histologically the freshly made fistulas produced in the horizontal semicircular canal by the two methods and found no discernible difference. The bony edges had the same appearance, with open blood vessels and lacunas and no evidence of the vessels' being filled with bone dust when the polishing burr was used. After the use of the burr there were more bone spicules left than after the use of the gouge.

The author then made defects in a cat's skull, using a rotary saw, a sharp burr, a sharp drill, a dull drill, a polishing stone and a polishing burr. Two weeks later the cat was killed and the skull sectioned. In each case the defects made with sharp instruments were filled with fibrous tissue containing many active osteoblasts producing new bone, whereas the defects made with dull instruments showed almost no fibrous tissue, no new bone and almost no osteoblasts. Since in each case the operative site was covered with periosteum, the author concludes that the difference in bone response was due to the different methods of producing the defects. The author believes that the type of tissue used to cover the defects is less important than the method used in making the defect when one wishes to create a permanently open fistula.

Perlman [23] contributes an interesting study of the process of healing in the labyrinthine capsule. He cites previous studies of available human material which have shown that in the first eleven days after a fracture of the labyrinth no evidence of healing can be made out, a fibrous tissue union can be seen after three weeks and a formation of new bone has been found at four weeks. However, no complete bony union occurred, since even after many years fibrous tissue was found uniting the fragments in various parts of the labyrinth. This helps to explain the clinical observation that acute otitis media occurring as long as sixteen years after a fracture of the labyrinth may result rapidly in fatal meningitis.

To add to these observations on the rather meager human material, the author examined histologically the labyrinths of rabbits and dogs at various periods after perforating injuries had been made in the cochleas with a needle. As in man, the labyrinth of the monkey, cat, dog and rabbit attains its maximum size before birth, and the striking absence of growth after birth is in marked contrast with what is observed in the other bones of the face. The blood supply of the capsule is distinct from that of the intralabyrinthine structures, and there are normally no anastomoses. The otic capsule in any of these animals, as in man, is composed of an outer periosteal layer with haversian canals formed from periosteum and a middle enchondral layer formed by osteogenic

23. Perlman, H. B.: Process of Healing in Injuries to the Capsule of the Labyrinth, Arch. Otolaryng. **29**:287 (Feb.) 1939.

buds, which excavate the early cartilage capsule, leaving islands of calcified cartilage. This enchondral bone remains the same throughout life, in contrast to similar bone elsewhere in the body, which is removed and replaced by haversian systems. An innermost endosteal layer of bone is formed in membrane.

The author used a needle to make perforating injuries in the cochleas. When such an injury was less than twelve hours old, blood cells were seen within the labyrinth, with fragments of bone at the site of perforation in part displaced outward, apparently by the escaping fluid. After one day the perforation appeared to be sealed by fibrin and blood cells. After three days the periosteum had swelled and proliferated, with fibroblasts replacing the blood clot, whereas the endosteal layer was unchanged. After six days there was new bone formation against the spicules of bone nearest the periosteal layer of the capsule, while activity in the endosteal and enchondral layers was lacking. After eight and ten days the formation of new bone was more marked and again was entirely from the periosteal layer. After seventeen days the perforation was largely filled with new bone, with a thick callus covering its site, and after twenty days the perforation was well healed with bone. After thirty and thirty-six days the callus seemed to be slightly swollen as the normal contour of the bone began to be restored.

The author notes that the principal factor in healing was the periosteal layer, while the endosteum shows little reaction of repair. Blood was found in the labyrinth as long as thirty days after injury and did not appear to act as an irritant. The organ of Corti, away from the perforation, remained normal. Infection, when present, was always associated with an accelerated healing, with earlier and more advanced formation of new bone. The new-formed bony plug which fills the perforation has its own blood supply. This acts as an anastomosis to the intralabyrinthine blood supply and thereby remains as a permanent pathway for vascular extension of infection from otitis media to the labyrinth. At the end of four weeks the activity of the tissue at the site of injury appears to have reached equilibrium. There is no tendency to progressive obliterative labyrinthitis from aseptic perforating injuries, such as is produced by suppurative processes in the labyrinth.

[These experimental studies are of fundamental importance in helping one to visualize what occurs in the human labyrinth when an artificial fistula is made. It is significant that in the presence of infection bone regeneration was accelerated. Of particular interest is the observation that after four weeks the activity of the tissue at the site of the injury appears to have reached an equilibrium. This suggests that the tendency to bony closure of a fistula is greatest the first few weeks. If a fistula remains open longer than four weeks, the tendency to bony closure diminishes and may eventually disappear.]

Šercer,[24] operating in 11 cases of advanced otosclerosis, used a modification of Sourdille's technic. With the region under local anesthesia, an atticoantrotomy was performed, with complete removal of the posterior and the superior bony meatus, leaving intact the membranous meatus. Removal of the incus and the head of the malleus was followed by cutting of a large skin flap from the membranous meatus, with the base of the flap at the upper margin of the drum. A fistula was then made in the lateral semicircular canal, covered with gold leaf and finally lined with the skin flap. The postaural wound was sutured. There have been no complications of note. In some cases there were striking improvements of brief duration; in other cases, results lasting more than two years. How to keep the fistula open is a biochemical problem which could be solved by finding the suitable metal for covering the fistula.

[The author is rather obscure concerning his exact results. His last statement implies that a satisfactory method for keeping the fistula open has not yet been found.]

Gasse[25] reports 14 cases of otosclerosis in which treatment was surgical. In 12 cases he employed Holmgren's technic, with the following results:

Case 1. No improvement in hearing, but tinnitus relieved, fifteen months after operation.

Case 2. No improvement in hearing, but tinnitus lessened, fourteen months after operation.

Case 3. No improvement in hearing twelve months after operation.

Case 4. No improvement in hearing six months after operation.

Case 5. Improvement in hearing from a 66 to a 60 per cent loss eight months after operation.

Case 6. Improvement in hearing from a 40 to a 33.3 per cent loss seventeen months after operation.

Case 7. Improvement in hearing from a 32 to a 26 per cent loss seven months after operation.

Case 8. No improvement in hearing.

Case 9. Improvement in hearing from a 36 to a 33 per cent loss five months after operation.

Case 10. Further loss, from 60 to 71 per cent. Tinnitus diminished.

Case 11. Improvement in hearing from a 64 to a 60 per cent loss four months postoperatively.

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24. Šercer, A.: My First Experiences with Operative Otosclerosis Therapy, Hals-, Nasen- u. Ohrenarzt (pt. 2) **49**:41 (July) 1939.

25. Gasse, E. R. G.: Surgical Treatment of Fourteen Cases of Otosclerosis, J. Laryng. & Otol. **54**:566 (Sept.) 1939.

Case 12. Improvement in hearing from a 68 to a 65 per cent loss three months postoperatively.

In 2 cases Lempert's technic was employed, with the following results:

Case 1. No improvement in hearing six months postoperatively. Tinnitus reduced.

Case 2. Further loss, from 32 to 51 per cent, three months postoperatively. Tinnitus lessened.

The author notes that while 5 of his patients showed audiometric improvement, only 3 had improved hearing for conversation. Tinnitus was reduced in 7 and completely relieved in 2. The fistula sign was present in all the patients whose hearing was improved and in 1 whose hearing decreased.

[The hearing improvements reported range from 3 per cent to a maximum of 6.5 per cent. Since the hearing in otosclerosis is considered to vary normally within a range of 10 decibels, or 8 per cent, it seems doubtful that the author's results can be regarded as significant.]

Hutchinson [26] presents an interesting report of a case in which a diagnosis of chronic adhesive deafness was made. An operation had been performed five years before with the use of Sourdille's technic. Examination of the hearing in February 1934 showed a bilateral conduction defect. Whisper could not be heard in either ear, and voice was heard only in the right ear. An audiometer was not available. Sourdille's operation was carried out in three stages on the left ear, in which the hearing was the poorer. Several years later, in May 1939, a whisper could be heard fairly well in the ear which had been operated on. An audiogram showed the hearing in this ear (the left) to be considerably better than that in the right ear, which had not been operated on:

Frequency	Decibel Loss on Left	Decibel Loss on Right
512	45	60
1024	40	55
2048	35	60

Two other patients on whom the author operated by the same technic experienced no permanent improvement in hearing. The author states that six to eight weeks is required to secure healing after each stage of the Sourdille procedure, while four to five months should be allowed to elapse between stages.

26. Hutchinson, C. A.: A Case of Membranous Periodic Deafness Treated Surgically by Sourdille's Procedure, J. Laryng. & Otol. **54**:714 (Dec.) 1939.

Lempert's procedure is referred to as being similar in many respects to Sourdille's operation. The author sees the following objections to it:

1. The exposure cannot be as free as by a postaural approach, and thus the difficulties are increased.

2. The fact that the eustachian tube is shut off at the same stage as the opening of the external semicircular canal must surely increase the risk of labyrinthine infection.

3. The trough-shaped fenestration would appear to increase the possibility of the flap's retracting from the bone, leaving a dead space which would interfere with proper vibrations of the new membrane.

4. Covering the fenestration with a tympanomeatal flap that has not become completely cicatrized increases the chance of contraction, leaving a dead space over the fenestra.

Finally the author points out that the majority of failures by early operators were due to infection and that the two or three stage procedure lessens the danger of infection in the final stage.

[Hutchinson's report is especially interesting because it contains the first and only audiometric record taken after the Sourdille procedure that has ever been published. Unfortunately, there was no preoperative audiogram. However, if one considers that the ear on which the operation was performed had the poorer hearing before operation, the audiometric record, showing that this ear now averages 18 decibels better for the conversational frequencies than the ear which was not operated on, indicates a substantial and significant hearing improvement five years after the Sourdille operation. Hutchinson's theoretic objections to the Lempert technic have not been borne out by experience.]

In a discussion of modern surgical procedures for operation on the labyrinth, following Hutchinson's presentation, W. M. Mollison [27] stated that he had seen only 1 of Sourdille's patients. This patient said that she heard better, but he could not detect improvement by his tests. Walter Howarth [28] had done 10 operations following Holmgren's technic or a modification of it, but in every case the hearing improvement, after twelve weeks in 1 case, suddenly disappeared. He expressed the belief that Holmgren's operation has not justified the claims made for it and says that as far as he can discover, Holmgren's late results were not very good, the hearing being only temporarily restored.

Hall [29] had done a number of operations by Holmgren's technic, with results similar to Howarth's. The problem seemed to be to keep

27. Mollison, W. M., in discussion on Modern Surgery of the Labyrinth, J. Laryng. & Otol. **54**:360 (June) 1939.

28. Howarth, W., in Discussion on Modern Surgery of the Labyrinth, J. Laryng. & Otol. **54**:363 (June) 1939.

29. Hall, in Discussion on Modern Surgery of the Labyrinth, J. Laryng. & Otol. **54**:365 (June) 1939.

the fistula open. Victor Negus [30] said that the operation is doomed to failure (by closure of the fistula by bone regeneration) unless an epithelial surface can be turned so as to face inward.

[It is interesting that Lempert uses Negus' suggestion in that he applies a Thiersch graft to the fistula with the outer surface facing inward, to prevent bone regeneration in revisions when bony closure has occurred.]

Kopetzky [31] presents sample cases in which different technics were employed to obtain fenestration of the labyrinth in patients with chronic deafness. In 2 cases the epitympanic space was not touched, the horizontal canal being exposed and fenestrated and covered with the skin of the posterior meatal wall. In the first case the ear which was operated on improved 8 per cent and that on which operation was not done improved 4 per cent, while in the second case the ear which was operated on improved 4 per cent and that which was not operated on improved 10 per cent. In another case the operation was done in two stages. The first stage consisted in exposure of the horizontal semicircular canal, removal of the head of the malleus and placing of the skin of the meatus over the unopened horizontal canal. In the second stage the skin was elevated from the horizontal canal, the canal was fenestrated, and the skin was then replaced. Five months after operation the ear which was operated on showed a gain of 25 per cent and that on which operation was not done, a gain of 11 per cent. The bone conduction, very low before operation, showed a marked gain, especially in the ear which was operated on. In another case the same operation was done in one stage, but fifteen days afterward the flap sloughed off and since then the fistula has been covered only with a blanket of granulations requiring continual cauterization with silver nitrate. In this case there has been a gain nine months after operation of 18 per cent in the hearing of the ear which was operated on and 13 per cent in that of the ear on which operation was not performed. In another case the same operation was done with removal of the incus. Six months after operation the ear which was operated on showed a gain of 32 per cent and that on which operation was not done, a gain of 7 per cent. In another case the membranous horizontal semicircular canal was destroyed. There was a slight gain, followed by a fall to the preoperative level, the function of the cochlea remaining unimpaired.

The author concludes from his experience that the material used to cover the fenestra is less important than the method used in making

30. Negus, V., in Discussion on Modern Surgery of the Labyrinth, J. Laryng. & Otol. **54**:366 (June) 1939.

31. Kopetzky, S. J.: Studies in Labyrinthine Fenestration to Improve Hearing: A Preliminary Report, Laryngoscope **49**:1064 (Nov.) 1939.

the fenestra. He believes that the heat produced by the polishing burr is an important factor in inhibiting the regeneration of bone. He also finds that a small fenestra gives better results than a large one. In some cases an initial gain in hearing has been lost, although the fenestra has remained open. Finally, the improvement which was observed in successful cases in the ear which was not operated on leads the author to believe that there is a reawakening of the cortical hearing centers.

[The author fails to give a complete account of his cases in which operations were performed, so that it is impossible to know his actual results. It is important that these be published, so that otologists may know which technic gives the greatest lasting hearing gain in the largest proportion of cases. Such improvement as was observed by the author in the ear which was not operated on has not been observed by me in 45 cases in which operation was performed by the Lempert technic. If the hearing in both ears improves, it seems likely that part or most of the improvement is the result of practice with the audiometer or is within the normal limits of variation. There should be several—if possible, four or five—preoperative audiograms at intervals of a week, and the average loss for all preoperative tests should then be taken as the preoperative hearing level. This will largely eliminate improvements from practice, and it will diminish errors due to normal variation.]

Wishart [32] presents a small instrument for removal of the head of the malleus in the Lempert fenestration operation. It consists of a small hook that passes in front of and behind the neck of the malleus, with a guillotine blade, which then severs the neck. The author believes that with the use of this instrument there is less tendency for the handle of the malleus to tear the membrana tympani and less danger of dislocating the incus than when the usual shears type of instrument is used.

Campbell [33] reports 8 cases in which operation was performed according to the technic of Lempert, with the following results: In 1 case the fistula test was moderately positive and there was a hearing improvement of 20 per cent eight months after operation. In a second case there was active nystagmus in the fistula test with an improvement of 24 per cent two months after operation. In a third case there was an active response in the fistula test with an improvement of 35 per cent five weeks after operation. In a fourth the fistula test was negative and the hearing gain lost three weeks after operation. Five months later the fistula was reopened, but within four weeks the initial gain in

32. Wishart, D. E. S.: Guillotine for Removal of Head of Malleus in Lempert Operation, Laryngoscope **49**:307 (April) 1939.

33. Campbell, E. H.: Experiences with Fistulization of the Labyrinth in Chronic Progressive Deafness, Arch. Otolaryng. **30**:689 (Nov.) 1939.

hearing had again been lost. In a fifth case the fistula test was negative and the hearing gain disappeared one month after operation. Four months later the fistula was reopened, and four weeks later the fistula test was positive and there was a hearing gain of 5 per cent. In 3 cases the postoperative hearing was considerably below the preoperative level. This was due in the first case to perforation of the membranous labyrinth and dislocation of the incus, in the second to a postoperative serous labyrinthitis and to absence of the drum membrane and ossicles because of a previous radical mastoidectomy and in the third case to perforation of the membranous labyrinth.

The author concludes that a definite improvement in hearing can be obtained in properly selected cases of conduction deafness by fistulization of the labyrinth according to the technic of Lempert. From his experience he emphasizes the importance of adhering strictly to Lempert's described technic if good results are to be effected. When the fistula closes by bone regeneration, the improvement is rapidly lost.

[This is the first published report of improvements in hearing secured by some one else using Lempert's technic. While far from perfect, these results surpass those secured in the treatment of otosclerosis by any previous author except Lempert. Campbell deserves commendation both for his audiometrically proved good results and for his complete report of results with no attempt to obscure or conceal errors and failures by omitting them from his report.]

Lempert [34] reports his results in 73 cases of otosclerosis. The patients are divided into two groups: 42 with good bone conduction and therefore suitable for operation and 31 with poor bone conduction, who requested that the operation be tried in the hope of some slight improvement.

Of the 42 suitable patients, 35 derived a high degree of permanent hearing improvement in the ear which was operated on. In 7 the hearing was not improved and remained at its preoperative level. Of the 31 unsuitable patients, 9 showed a good improvement, 20 had hearing at the preoperative level and 2 showed a further loss.

Of the entire 73 patients operated on, the fenestra remained permanently open in 62 and closed in only 11. Revision of the operative site in these 11 showed new bone formation in 9 and fibrotic occlusion of the fenestra in 2. In the vast majority tinnitus was greatly diminished or had disappeared.

The author emphasizes the proper selection of patients for operation and the importance of perfection and exactitude in carrying out every detail of the technic. The membrane used to close the fenestra must

34. Lempert, J.: Improvement of Hearing in Otosclerosis: Report of Seventy-Three Cases, Ann. Otol., Rhin. & Laryng. **48**:544 (June) 1939.

be the thinnest possible and the most vibratory, so as to interfere as little as possible with the mobility of the perilymph.

In fairness to the already successful development of surgical treatment for otosclerosis and for the protection of its still brighter future the author states that no otologist, no matter how skilful, should attempt this particular operation without receiving special training in this type of surgery under supervision and guidance.

[The author's term "permanent hearing improvement" should be replaced by "hearing improvement lasting so many months," since too brief a period has elapsed for one to know the ultimate fate of any hearing improvement after fenestration. It is true that when the hearing gain has been maintained for three months or more the tendency toward bony closure of the fistula with loss of the hearing improvement seems to be greatly diminished, but it is not yet known for certain how late a fistula can close or whether the nerve degeneration that sooner or later occurs to a varying degree in all cases of otosclerosis will eventually destroy the hearing gained by the operation. However, even should nerve degeneration nullify the effects of the procedure five to ten years later, the operation would still be worth while. Lempert's brilliant results have not been approached by otologists using any other technic. Until better results are reported obtained by some other technic, Lempert's procedure must be regarded as the only operation which at present can promise a satisfactory hearing gain in a majority of properly selected cases. There is little doubt that the results secured will be in direct proportion to the skill and care with which the intricate and difficult technic devised by Lempert is carried out in its minutest detail and that otologists who attempt this operation without first mastering every detail of the technic on cadavers will fail to secure permanent results. The fault will lie not so much with the operation as with the operator.]

LABYRINTHINE DEAFNESS

Pathology.—Lurie [35] exposed animals to loud sounds and to overdosage with quinine and observed degeneration of the external hair cells before there was visible damage to the internal hair cells. Correlating this observation with the anatomic studies of Lorente de Nó, who found that a single nerve innervates one or two internal hair cells at most while a nerve to the external hair cells innervates a large number, having been traced for a quarter turn of the cochlea, the author concludes that the external hair cells are probably concerned with sounds of small intensity and the internal hair cells with pitch. It has been

35. Lurie, M. H.: What Is Perception Deafness from a Physiological and Histological Basis? Ann. Otol., Rhin. & Laryng. **48**:3 (March) 1939.

found in confirmation of this theory that discrimination of pitch is more accurate for loud sounds. Another point in favor of this theory is the fact that the external hair cells always rest on the basilar membrane, which presumably vibrates with sounds of slight intensity, while the internal hair cells may rest on the bone of the lamina spiralis ossea, which presumably vibrates only with loud sounds. Further evidence in support of the theory is found in the fact that prolonged loud sounds cause degeneration of the external hair cells, due presumably to very strong vibrations of the basilar membrane, while the internal hair cells, resting on bone, remain intact.

Because histologic changes in the stria vascularis have been observed with normal hearing, the author does not believe that changes in the stria can cause deafness.

From Japan comes a number of contributions on experimentally produced changes in the inner ear.

Iwata [36] produced secondary, hemolytic and aplastic anemias and studied the histologic and clinical effects. In acute loss of blood there is only a transitory decrease in hearing, with no histologic changes. In long-standing anemias Preyer's aural reflex is diminished, nystagmus after rotation is diminished and histologically there is degenerative atrophy of the spiral ganglion, of Corti's organ, of the stria vascularis and of the ligamentum spirale cochleae as well as degeneration of the cochlear and the vestibular nerve. Finally, atrophy and halisteresis of the bone occur.

Senoo [37] produced epinephrine arteriosclerosis by giving epinephrine, vigantol (a preparation of irradiated ergosterol) and calcium, and cholesterol arteriosclerosis by giving egg yolk. The histologic changes observed in the inner ear were due, he states, not to the arteriosclerosis but to the toxic effect of these substances, with advanced degenerative atrophy of all parts of the cochlea, especially of Corti's organ, and diminution or loss of the vas spirale. The atrophy was always directly proportional to the amount of the substances administered. There was little degenerative change in the vestibular apparatus.

Torii,[38] having found diminished hearing for low and middle tones with less involvement of the high tones and slight disturbances of the vestibular apparatus in the beginning of pregnancy, produced condi-

36. Iwata, I.: Concerning Disturbances of the Auditory Organ in Anemia, Ztschr. f. Oto-, Rhin- u. Laryng. 1938, vol. 44; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **32**:624, 1939.

37. Senoo, H.: Concerning Changes in the Auditory Organ in Experimental Arteriosclerosis, Ztschr. f. Oto-, Rhin- u. Laryng. **44**:51, 1938; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **32**:625, 1939.

38. Torii, G.: Concerning Impaired Hearing in Pregnancy and the Puerperium, Ztschr. f. Oto-, Rhin- u. Laryng. **44**:175, 1938; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **32**:668, 1939.

tions resembling those of pregnancy in rats by injecting an anterior pituitary extract containing the luteinizing factor, the urine of pregnant women and the urine of women after normal parturition. He observed diminution of glycogen and increase in fatty substance in the auditory organs, with atrophy of the spiral ganglion cells in the apical coil.

Sato [39] observed that after overdosage with vigantol (irradiated ergosterol) there was degeneration of the ganglion cells of the cochlear nerve as well as both destructive and productive processes in the bony labyrinth.

[It is difficult to see the clinical significance of these Japanese contributions except as confirmation of what has previously been known, i. e., that the eighth nerve may be injured by various toxic substances.]

Carnevale [40] examined histologically the middle and inner ears of a woman of 92, who died of senile decay and had never suffered from an aural disease. He describes the findings in detail, the most important of which was involution of the organ of Corti. He failed to find deposits of lime in the basilar membrane, as described by Mayer, but he observed lipoid-like deposits in the cristae and in the macula acustica and connective tissue formation in the lumen of the semicircular canal.

Hereditary Nerve Deafness.—Uffenorde [41] examined 203 deaf-mutes and decided that in 46 the condition was hereditary, in 55 it was acquired and in 102 the cause was uncertain. Marked disturbances in pneumatization were found in 27 per cent of the cases in which the condition was hereditary and in 50 per cent of those in which it was acquired. The larger incidence of disturbed pneumatization in the persons with acquired deaf-mutism is evidence of inferiority of the mucous membrane, which results in an increased susceptibility to continued inflammation.

Traumtaic Nerve Deafness.—An interesting article on deafness in aviators is contributed by Dickson, Ewing and Littler.[42] The authors state that the loudness levels produced by airplane engines are higher than those of the noise in boilermakers' shops. In aviators who fly without helmets in enclosed cockpits, there is a persistent loss of hearing after even a few hundred hours of flying, evidenced first by an abrupt

39. Sato, M.: Concerning Changes in the Eighth Nerve Through Overdosage with Vigantol, Ztschr. f. Oto-, Rhin- u. Laryng. **44**:33, 1938; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **32**:630, 1939.

40. Carnevale, R.: Anatomic-Histological Contribution to the Study of Senility of the Auditory Organ, Arch. ital. di otol. **5**:51, 1939.

41. Uffenorde, H.: The Significance of X-Ray Pictures for the Diagnosis of Hereditary Deafness, Hals-, Nasen- u. Ohrenarzt (pt. 1) **30**:88 (March) 1939.

42. Dickson, F. D. D.; Ewing, A. W. G., and Littler, T. S.: The Effects of Aeroplane Noise on the Auditory Acuity of Aviators: Some Preliminary Remarks, J. Laryng. & Otol. **54**:531 (Sept.) 1939.

dip near 4096. This defect is progressive so long as the person continues flying or running very noisy aircraft. Aviators' deafness must be considered, therefore, as threatening a serious and life-long disability.

The authors flew with unprotected ears for one hour in a twin-engined bomber, in which the noise level was 119 phons (B.S.), much below the maximum level reached in modern aircraft. Ten minutes after they landed audiograms showed:

1. The ear nearer the engine was the deafer.

2. Tinnitus in this ear, lasting nearly three hours, corresponded to about the pitch of 4000 double vibrations.

3. The audiograms showed the characteristic dip at 4096 and 8192, with no loss for other tones.

4. The bone conduction fell with the air conduction.

5. Recovery of hearing took place within an hour.

The authors cite Stevens and Davis, who observed in animals a physiologic phenomenon which they called hysteresis, or overloading—as the intensity of the sound increases, the response finally begins to decrease. They attribute this depression in response to a physiologic insult, from which recovery occurs after a few hours. Still greater intensities cause disruptive and degenerative changes in the hair cells, from which recovery may be only partial.

Experimentally, a considerable variation in susceptibility to auditory fatigue has been found in different persons.

The authors analyzed the pitch of airplane noise and found that the tones that were loudest were invariably between 30 and 200 vibrations, while in no case were tones present above 4000. The fact that these loud low tones always result in high tone deafness needs explanation.

The authors found that packing the canal with liquid petrolatum and with cotton saturated with petrolatum gave the maximum protection to the ear (equal to 50 decibels for sounds of 4000 vibrations, 25 decibels for 800 to 2000 vibrations and 15 decibels for 250 vibrations). A flying helmet gave somewhat less protection, amounting to 35 decibels for 4000 vibrations, 25 decibels for 2000, 15 for 800 and 5 for 250 vibrations. The authors conclude that protection of the ears appears to diminish, if not to eliminate, the risk of hearing impairment from noise.

[The information gained from aviators, that permanent damage to the hearing can be prevented by plugging the canals or by wearing a helmet, should be applied to industry, where no concerted effort has yet been made to prevent occupational traumatic deafness in boilermakers, pneumatic riveters and others who must work in a noisy environment.]

Larson [43] studied 250 laborers in shipyards or machine factories. Of the total number, 127 were excluded as having deafness due to something other than noise, leaving 123, of whom 64.4 per cent showed a reduction in hearing for whisper to a distance of less than 8 M. Both the proportion of deaf persons and the degree of deafness increased considerably with advancing age and increasing duration of work. Thus, not a single laborer above the age of 51 could hear whisper at more than 8 M., and after thirty-one years of work the hearing was always less than 1 M. for whisper. Tinnitus was present in 52 per cent, but there was no relation between the tinnitus and the deafness. Vertigo was present in only 5. Audiograms showed in many a circumscribed defect around 4096, such as was first described by Shambaugh and later by Bunch as a characteristic finding in professional deafness. Great variations in the deafness were found in some persons, depending on whether the hearing was tested immediately after work or after periods of idleness. The presence of middle ear suppuration did not appear to increase the amount of occupational deafness. The author believes that the characteristic dip near 4096 is due to a special vulnerability of this part of the cochlea, since the greatest intensity of the noises to which these men had been exposed lay not at this level but between 1000 and 2000 vibrations.

The prophylaxis of occupational deafness consists above all in reduction of the noise. Closure of the auditory canal is considered of little value by the laborers and by the author. If possible, persons suffering from inner ear deafness should be excluded from noisy occupations.

[The author's statement that closure of the meatus is of little value in the prevention of occupational deafness is contrary to the experience of aviators as reported in the previous article.]

Nerve Deafness from Infections.—In a discussion on inner ear deafness Watkyn-Thomas [44] emphasizes the importance of blood-borne toxins from focal infection, the teeth, especially infected wisdom teeth and crowned stumps, being the most important sites of infection. Tonsils are a bad second source. Sinusitis with retention of pus is uncommon as a cause. The gallbladder, appendix and prostate must be kept in mind as possible foci. The author mentions the use of roentgen examination of the labyrinth to confirm the clinical diagnosis of primary cochlear otosclerosis.

[I know of no description of any changes in cochlear otosclerosis which are to be observed on roentgen examination and doubt that the

43. Larson, B.: Investigations of Professional Deafness in Shipyard and Machine Factory Labourers, Acta oto-laryng., 1939, supp. 36.

44. Watkyn-Thomas, P. W., in Discussion on Internal Ear Deafness, J. Laryng. & Otol. **54**:273 (May) 1939.

roentgen ray would be able to show any recognizable changes caused by the disease.]

Goodhill [45] presents a valuable study of the temporal bones of 8 adults with syphilis and 8 infants with prenatal syphilis. The following changes observed histologically were quite constant:

1. Productive periostitis producing invasive fibrosis, followed by ossification of the perilymphatic and the endolymphatic channels of the vestibular apparatus, with malformation of the bony canals.

2. Bony abnormalities of the stapes, especially of the footplate.

3. Atrophy of the neuroepithelial elements of the cochlear and the vestibular system.

4. Lymphocytic infiltration of the cochlea and the spiral ganglion (miliary gummas).

5. Vascular changes, consisting of diapedesis in infants with prenatal syphilis and obliterative endarteritis in adults who had acquired the disease.

The author finds no relationship between syphilis and otosclerosis. One of his patients showed both conditions in the same ear, but the histologic changes observed in connection with each disease were distinct and different, so the association must be regarded as coincidental and not causal.

It is of interest that audiograms of 3 of the patients, taken shortly before death, showed practically normal hearing in 2, 1 with round cell infiltration throughout the membranous cochlea and 1 with atrophy of the organ of Corti and the spiral ganglion cells, while in the third a moderate low tone loss was found, but histologically the middle ear and the cochlea appeared normal.

Nerve Deafness from Avitaminosis.—Selfridge [46] continues to be interested in vitamins as related to nerve deafness. Because deficiencies of various vitamins, including C, B and especially the B_2 complex, have been found to produce degeneration of the eighth nerve in animals, the author has employed large doses of vitamins with correction of unbalanced diets in cases of nerve deafness. Reports are presented of 6 cases, in which nicotinic acid was given for months at a time, resulting in definite audiometric improvements averaging 5 to 10 decibels throughout the tone range but in some cases amounting to 15 to 25 decibels for the lower tones. The author concludes that while the entire story of the significance of the various functions of the B_2 complex has not yet been written, his observations suggest the importance of nicotinic acid in nerve deafness.

45. Goodhill, V.: Syphilis of the Ear: A Histopathologic Study, Ann. Otol., Rhin. & Laryng. **48**:676 (Sept.) 1939.

46. Selfridge, G.: Nicotinic Acid and the Eighth Nerve, Ann. Otol., Rhin. & Laryng. **48**:39 (March) 1939.

In his second article Selfridge [47] reports 6 cases of nerve deafness, in which treatment was with injections of riboflavin, thiamine hydrochloride or nicotinic acid. In the first patient, a man of 39, riboflavin produced no improvement, nicotinic acid produced a definite improvement in both ears and thiamine hydrochloride produced no further improvement. In the second, a woman of 43, nicotinc acid produced an improvement in hearing in both ears and thiamine hydrochloride resulted in an additional improvement. In the third, a man of 59, nicotinic acid produced a gain chiefly in one ear, while thiamine hydrochloride produced a gain chiefly in the other ear. In patient 4, a man of 60, nicotinic acid produced an improvement, and further improvement occurred with the administration of thiamine hydrochloride. In patient 5, a man of 65, nicotinic acid produced a definite improvement, with a further gain in one ear after the administration of thiamine hydrochloride. In patient 6, a man of 73, nicotinc acid and riboflavin produced a slight gain, thiamine hydrochloride produced no further gain and continued use of nicotinic acid resulted in additional improvement.

The author concludes that thiamine hydrochloride is an essential addition to nicotinic acid but that the greatest improvement occurs from the use of nicotinc acid. The data submitted suggest strongly to him that nutritional deficiencies offer the best explanation of the degenerative processes involving the eighth nerve.

In his final article Selfridge [48] reports the case of a child of 9 with unilateral high tone deafness of three years' duration following active otitis media. With the patient on a high vitamin diet plus the vitamin B complex, carotene and vitamin C there was a slight audiometric improvement. A girl of 18 who had had unilateral tinnitus and deafness for eighteen months was given vitamin C, thiamine hydrochloride and nicotinic acid, with a striking improvement in the tinnitus and deafness. Other cases reported are evidently the same ones reported in the preceding article.

[The strongest argument against the theory that vitamins are important in nerve deafness is the fact that deafness is not a symptom of clinical avitaminosis; neither scurvy, beriberi nor pellagra have been shown to be associated with nerve deafness. If marked clinical avitaminosis does not injure the eighth nerve, it is difficult to believe that mild subclinical avitaminosis would be a cause of nerve deafness. While not convinced that vitamins are a factor in nerve deafness, I have attempted to maintain an open mind and have employed them in numerous cases of nerve deafness, with uniformly negative results to date.]

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47. Selfridge, G.: The Eighth Nerve in Relation to Thiamin Chloride and Nicotinic Acid, Ann. Otol., Rhin. & Laryng. **48**:419 (June) 1939.

48 Selfridge, G.: Eighth Nerve Deafness from a Nutritional Standpoint, Ann. Otol., Rhin. & Laryng. **48**:608 (Sept.) 1939.

Adam [49] describes a case in which Ménière's syndrome was apparently due to avitaminosis. A woman of 36 suffered from attacks of vertigo and unilateral deafness of three years' duration. Because of a deficiency in her diet and an absence of ascorbic acid in the urine in three successive tests, she was given fresh fruits, cod liver oil and vitamin B complex. The attacks of vertigo disappeared within two months, the hearing became normal and the patient has remained free from her former symptoms for eighteen months. In 4 other cases Ménière's disease was found associated with a deficient intake of vitamin C and was cured largely by attention to vitamins.

Mellanby [50] makes some interesting observations on young dogs fed on diets deficient in vitamin A but rich in all other vitamins. The dogs showed evidence of deafness during life. Histologically there was degeneration of the spiral ganglion and of its central and peripheral branches to the point of complete disappearance. The vestibular portion of the eighth nerve showed degeneration, but always to a distinctly lesser degree. Overgrowth of bone in the modiolus and of bone around the internal auditory meatus was a constant finding and led to pressure on and stretching of the eighth nerve in the meatus. The degeneration of the cochlear nerve apparently was due to this pressure, with secondary degeneration of the spiral ganglion cells. The degenerative reactions of the cochlear nerve thus represent a notable exception to Waller's law. Cutting of the vestibular nerve, however, does not lead to degeneration of Scarpa's ganglion. This difference has been noted by other workers previously (Wittmaack; Hallpike; Crowe). Degeneration of the optic and the trigeminal nerve in dogs used for the experiment seems to have been due to the same overgrowth of bone in the skull with pressure on these nerves.

A second finding in these dogs on a diet deficient in vitamin A was serous labyrinthitis in the cochlea, resulting in degeneration of the sensory epithelium of the labyrinth, which extended in the course of time to the epithelium of the organ of Corti and to that of the ampullae of the semicircular canals.

[One must be cautious in applying the results of these animal experiments to human beings. For one thing, the bony changes observed in dogs have never been described in human beings. Second, complete absence of vitamin A from the diet of human beings practically does not occur, though partial deficiencies undoubtedly do.]

49. Adam, J.: Ménière's Syndrome and Avitaminosis, J. Laryng. & Otol. **54**: 256 (May) 1939.

50. Mellanby, E.: The Experimental Production of Deafness in Young Animals by Diet, Laryngoscope **49**:1090 (Nov.) 1939.

Nerve Deafness from Miscellaneous Causes.—Kelley [51] tested 88 subjects from 50 to 86 years of age audiometrically for tone discrimination and for perception of speech. The audiograms showed progressive loss for high tones. From 50 to 59 years of age there was an average loss of 15 decibels for 2048, from 60 to 69 there was a 25 decibel loss for this tone and from 70 to 99 there was a beginning loss for 1024, averaging 15 decibels. Hearing for low tones was not affected until the subject was above 70 years, when it began to show a slight impairment.

A person over 60 does not recognize the elimination of overtones above 4000 vibrations from a violin tone. A person over 60 has no difficulty with vowels but recognizes only 75 per cent of consonants at ordinary levels of intensity for conversation. This is accounted for by the high tone loss, since consonants contain high frequency components.

[The author neglects to add that consonants have less intensity than vowels and are therefore more poorly heard when there is any hearing loss.]

Treatment of Nerve Deafness.—Koch [52] reports 19 cases of presbycusis in which treatment was with testosterone (proviron) and estradiol benzoate (progynon B), given intramuscularly in most cases, by mouth in some. He emphasizes that deafness due to other causes than low levels of these substances must be excluded. The dosage varies individually, with large doses being given at the beginning, the amounts being slowly decreased. The treatment resulted in disappearance of tinnitus and marked improvement in hearing lasting eighteen months to two years or longer. There were no bad effects.

[It is difficult to understand how there can be "marked improvements in hearing" in a degenerative process such as presbycusis. Since the author does not substantiate his results audiometrically, they cannot be accepted as proved.]

Hayden and Chainski [53] discuss the causes and treatment of tinnitus. They stress the importance of determining the cause and, when possible, of eliminating it. When the cause cannot be eliminated, various medicaments may be employed. These include tincture of cimicifuga racemosa, 15 drops, increased to 60 drops a day; ephedrine, 0.5 grain (0.03 Gm.) three times a day, combined with a proprietary belladonna

51. Kelley, M. H.: A Study in Presbyacusis, Arch. Otolaryng. **29**:506 (March) 1939.

52. Koch, F. X.: Prospects and Treatment of Presbyacusis, Ztschr. f. ärztl. Fortbild. **36**:392 (July 1) 1939.

53. Hayden, D. B., and Chainski, E. L.: Tinnitus: Etiology and Evaluation of the Various Methods of Treatment, Ann. Otol., Rhin. & Laryng. **48**:433 (June) 1939.

preparation (bellafoline), 0.25 mg. to 1 or 2 mg. a day, and bromides and barbiturates. Surgical measures that may be resorted to in the most distressing cases are cervical sympathectomy and section of the eighth nerve.

Ménière's Syndrome.—Hallpike [54] adds a histologic study of a third case of Ménière's disease to those of the 2 previously reported by him. In January 1938 a man of 29 began to notice a defect in hearing and a rushing tinnitus and sense of fulness in the left ear. These symptoms continued intermittently until April, when he had a sudden severe attack of vertigo with vomiting. Similar attacks recurred every two or three weeks until he was admitted to the hospital in September. For some years he had been troubled with nasal obstruction and a thick discharge, with a tendency toward attacks of sneezing and running of the nose. For some months there had been stiffness and weakness of the joints. Hearing tests showed a considerable loss in the left ear, more for high tones, with shortened bone conduction, and normal hearing on the right. The drum membranes were normal. The tonsils were enlarged and infected. Roentgen examination showed bilateral antral sinusitis. On September 19 tonsillectomy and bilateral intranasal antrotomy with submucous resection of the septum were done. Nine days later a secondary hemorrhage began, and in spite of a transfusion and numerous intranasal packs death occurred October 29 from repeated hemorrhages. Injection of the tympanic membranes, due doubtless to the nasal packs, was noted two weeks before death, but no discharge from the ears occurred. The temporal bones were removed twenty hours after death.

The right temporal bone was normal except for pus in the tympanic cavity and in the mastoid cells, without osseous changes. Except for postmortem changes, the right membranous labyrinth appeared normal.

The left temporal bone showed the same changes as the right in the middle ear spaces. In the left labyrinth there was considerable distention of the scala, with Reissner's membrane thrown back into contact with the wall of the scala vestibuli over a third of its length. A number of folds in Reissner's membrane were evidence of a considerably greater distention of the scala media during life. The left saccus endolymphaticus was tightly packed with a red-staining colloid material, and there was absence of the normal loose-meshed connective tissue around the saccus. Examination of the eighth nerves did not show any abnormalities. The organ of Corti and the sensory cells of the maculas of the semicircular canals appeared normal except for postmortem changes.

54. Hallpike, C. S.: On the Histological Changes in the Temporal Bone of a Case of Ménière's Disease, Proc. Roy. Soc. Med. **32**:1646 (Oct.) 1939.

Yamakawa [55] adds a fourth case of Ménière's disease in which histologic examination was done to the 3 described by Hallpike. The patient had had many attacks of vertigo, tinnitus and deafness of varied duration. Histologically the inner ear showed bulging of Reissner's membrane into the scala vestibuli in all turns and the formation of concrements in the stria vascularis and in the aqueduct of the cochlea as well as in the internal auditory meatus. The lumen of the endolymphatic duct and of the saccus endolymphaticus on the affected side was narrow compared with that on the other side and was filled with a thick colloid mass. Corti's organ, however, including the nerve cells of the spiral ganglion and of the vestibular ganglion, was intact. There were a few albuminous coagulations in the cochlear duct. The author explains the attacks on the basis of the findings as follows: The secretion of the stria vascularis was disturbed by the concrements. This resulted in irritation, which was manifest in attacks by which the pressure in the endolymph space was raised and the function of the sensory cells was disturbed. By diffusion and resorption of the endolymph and stretching of Reissner's membrane, the raised pressure was leveled and the sensory cells recovered their function. As the coagulum in the cochlear duct and the colloid material in the endolymphatic duct and sac showed, the increased endolymph became thicker. The cause of the formation of the concrements is not clear.

[The extraordinary similarity of the findings in all 4 cases of Ménière's disease that have been studied histologically is striking. These findings are essentially a marked dilatation of the ductus cochlearis, with displacement of Reissner's membrane, and usually the presence of some albumin or colloid material in the cochlear duct or in the ductus or saccus endolymphaticus. The essential pathologic lesion in Ménière's disease is thus an abnormal amount of endolymph with probably some abnormality of its constitution, as evidenced by the pink-staining coagula and the colloid material. Several possible mechanisms suggest themselves: an inflammatory condition resulting in oversecretion of endolymph and accumulation of serum mixed with endolymph; an allergic condition with edema and accumulation of fluid; a primary disease of unknown cause affecting the secreting cells of the stria vascularis or the absorbing cells of the saccus endolymphaticus. Clinical experience indicates that the first two mechanisms probably account for many of those cases of Ménière's disease in which removal of a focus of infection or of an allergic factor is followed by disappearance of the symptoms and by improvement and even recovery of the hearing. The work of Mygind and Dederding and of Furstenberg, who showed

55. Yamakawa, K.: Concerning Pathologic Changes in a Ménière Patient, Ztschr. f. Oto-, Rhin- u. Laryng. **44**:181, 1938; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **32**:697, 1939.

that retention of fluid increases the symptoms, is in harmony with the view that abnormal accumulations of endolymph are responsible for the attacks. Anything that diminishes the tendency to edema, whether it be limitation of the intake of sodium or of the total intake of fluids, elimination of an allergic factor or removal of a focus of infection, will result in improvement.]

Hallpike [56] reviews the histologic findings in Ménière's disease, i. e., a very marked dilatation of the scala media in the cochlea, so that Reissner's membrane was displaced upward to obliterate entirely the scala vestibuli and degeneration of the cells of Corti's organ in 2 cases and of the cells of the cristae of the semicircular canals in another. The normal loose perisaccular connective tissue was lacking in all cases, suggesting an impairment in the resorption of endolymph. However, since this condition was also found in 2 of 13 normal labyrinths, it does not explain the symptoms and other findings. The author concludes that there must have been also hypersecretion of endolymph, resulting in marked dilatation of the scala media. This dilatation was so extreme as to obliterate the scala vestibuli, and thus the mechanism for equalizing the pressure between endolymph and perilymph furnished by Reissner's membrane was eliminated. Any further secretion of endolymph would lead to sudden and marked increases in pressure, with asphyxia of the sensory cells and sudden stimulation of these cells, producing vertigo. The author does not hazard a guess as to what causes the hypersecretion of endolymph.

Dohlman [57] reports 4 cases of Ménière's disease caused by a metabolic disturbance. In the first case eczema of the hands, attacks of vertigo, vomiting and eosinophilia disappeared after milk was omitted from the diet. In the second a flour allergy with attacks of Ménière's disease was cured with a wheatless diet and avoidance of contact with flour. In the third the patient was cured with general hygienic treatment (diet and mode of life) and in the fourth by a diet suited to the patient's digestion. In the last 2 cases there was no evident hypersensitivity.

Skoog [58] studied the "carotid syndrome" produced in the guinea pig by injecting sheep cell–hemolytic rabbit serum into the right carotid artery, toward the aorta. When the serum has been injected, the animal assumes a pronounced flexion of the whole body toward the left, with deviation of the right eye anteriorly toward the nose and the left eye posteriorly toward the ear. There is no spontaneous nystag-

56. Hallpike, C. S.: Some Recent Work on Intralabyrinthine Pressure, Acta oto-laryng. **27**:229, 1939.

57. Dohlman, G.: Ménière's Disease on Allergic Basis, Acta oto-laryng. **27**:245, 1939.

58. Skoog, T.: Studies of a Vestibular Syndrome Induced in Guinea Pigs by Allergic Reaction, Acta oto-laryng., 1939, supp. 32, p. 1.

mus. After ten minutes the animal moves constantly in a circle toward the left. After fifteen minutes there is slight spontaneous nystagmus. After five hours the animal is normal in all respects.

Study of the brain of the affected animal by means of acid dyes intravitally introduced reveals marked staining in the medullopontile region on the side of the injection, indicating an allergic breakdown of the capillary walls in this region. The author concludes that the vestibular syndrome observed results when the injected serum reaches the brain stem via the vertebral artery and there evokes an allergic reaction of the capillaries. The author states that his findings do not in any way exclude the possibility that in man the peripheral labyrinth participates in some other way, since one may well conceive of allergically sensitive tissues also being present in the labyrinth.

[All histologic evidence, as well as the clinical evidence, indicates that in man the labyrinth is the organ affected in Ménière's disease. The finding of diplacusis, which in my experience is always present in Ménière's disease when the hearing is affected, is, I believe, conclusive proof of involvement of the membranous labyrinth by an edema, since diplacusis cannot be produced by a lesion of the nerve or of the central pathways.]

Mygind and Dederding [59] continue their studies of water balance in Ménière's disease, noting that after a diuretic is given there may be an improvement in the hearing and general condition of the patient, whereas an intake of large amounts of water (800 cc.) may result in further loss of hearing, nystagmus and headache without effect on the heart or kidneys. They studied histologically, in animals, the effects of disturbance of water balance on the labyrinth. Reissner's membrane was bulged out, a sign of increased pressure in the ductus cochlearis. The cells of the organ of Corti were swollen and drops of free hyalin appeared in them. After injections of a solution of sodium chloride the cochlear duct and Corti's organ shrank. The same reactions occurred after the administration of pilocarpine or atropine, presumably due to diminution in the secretion of endolymph with reduction in endolymphatic pressure.

The authors explain the striking influence of fluctuations of water metabolism on the labyrinth as due to the peculiar anatomic conditions. In a closed hydrostatic system slight oscillations of volume can cause remarkable oscillations of pressure. The nystagmus, the disturbances in sound conduction, the fluctuations in bone conduction, the occasional fistulous symptoms, the nystagmus on catheterization of the tubes, the unpleasant sensation of pressure in the ear, all can be explained by increased pressure in the labyrinth.

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59. Mygind, S. H., and Dederding, D.: Clinical and Experimental Studies of Ménière's Disease, Orvosi hetil. **83**:77 (Jan. 28) 1939.

The authors conclude that Ménière's disease is not a specific disease but is a complex of symptoms caused by different etiologic factors, including heredity, infection, hormonal influences, dietary mistakes and bodily and especially mental strain. Fundamentals of therapy are removal of water from the body, improvement of circulation, local treatment of the ears and removal of causes of the disease.

[The observations on animals, corresponding to Hallpike's histologic studies on human beings, are especially interesting.]

Woodman [60] reports 11 patients with vertigo, tinnitus and deafness who were operated on by the Portmann procedure: The mastoid process is opened and the bone removed down to the dura of the posterior fossa in front of the lateral sinus. The dura is then carefully lifted from the posterior-superior surface of the petrous bone. The point where the ductus vestibuli reaches the surface is carefully noted and is marked by a small adhesion. This is forcibly elevated, and in favorable cases a small drop of clear fluid is seen to come out. If the aqueduct is not located, the dura is elevated until the internal auditory meatus is reached, by which time it is clear that the duct must have been passed. The parts are allowed to fall together, and the wound is sutured without drainage. The difficulties encountered in the operation are extreme density of the bone, a forward lying lateral sinus and hemorrhage from the emissary vein. An absolute contraindication is infection in the middle ear spaces.

Of the 11 patients operated on, 8 were completely cured of vertigo, 1 became much better, 1 cannot be traced and for 1 the operation failed. In the last an atresia of the duct was suspected. The tinnitus was completely cured in 2, greatly improved in 1 and unchanged in 8. The hearing was practically restored to the normal in 1, improved in 5, unchanged in 4 and worse in 1.

The author concludes that the Portmann operation was preferable to section of the eighth nerve because of greater safety and better chance of improved hearing and preferable to the injection of alcohol into the labyrinth, because the latter procedure destroys the hearing.

[Portmann's original contention that Ménière's disease is due to hypertension of the endolymph appears to be borne out by the work of Hallpike and of Mygind and Dederding.]

Brunner [61] defines Ménière's syndrome as attacks of tinnitus and deafness, dizziness, spontaneous nystagmus, diminished excitability of one labyrinth and general vasomotor symptoms. It is possible that one or another of these symptoms may be missing in an attack, in which event further observation is required to make the diagnosis definite.

60. Woodman, E. M.: The Position of the Portmann Operation in Relation to Labyrinthine Vertigo, Proc. Roy. Soc. Med. **32**:1642 (Oct.) 1939.

61. Brunner, H.: Ménière's Syndrome, Laryngoscope **49**:877 (Oct.) 1939.

Symptomatic Ménière's syndrome, in which the attacks are a symptom of a disease such as leukemia, or of injuries to the head, arteriosclerosis of the brain, acoustic neuroma, Paget's disease of the temporal bone, tabes, syphilitic neurolabyrinthitis, chronic adhesive otitis media or otosclerosis, is distinguished by the author from idiopathic Ménière's disease, of which the cause is unknown. The author believes that idiopathic Ménière's syndrome is due to attacks of serous labyrinthitis with serous and cellular exudate in the perilymph and to a less extent in the endolymph and with abnormal pressure in the intralabyrinthine fluids due to obstruction of the cochlear aqueduct by the exudate. The serous labyrinthitis may be due to osteitis of the labyrinthine capsule adjacent to the labyrinth (Paget's disease) or to focal infection. The author compares serous labyrinthitis with vasomotor rhinitis and mentions the finding of allergy as an etiologic factor by certain authors.

The attacks of vertigo may cease spontaneously. The tinnitus is less likely to stop, and the hearing usually remains impaired. The treatment of idiopathic Ménière's syndrome should first be conservative: dietary measures, as recommended by Mygind and Dederding and by Furstenberg; removal of foci of infection, as recommended by Wright, and faradization of the ear and inflation by means of a catheter, as recommended by the author. Surgical section of the vestibular nerve should be reserved for the few patients with severe unilateral deafness that defies all efforts at conservative treatment.

[This is an excellent review of the subject, with a logical therapeutic approach. The author neglects, however, the eliminating of any food allergy, which, in my experience and in that of Dean and Linton, of Jones and of Dohlman, is a not infrequent cause of Ménière's syndrome.]

Von Bárány [62] considers monotrean (a quinine-papaverine preparation) the most effective drug for the treatment of Ménière's syndrome. This drug, a combination of quinine and papaverine hydrochloride, seems to have a selective action on the labyrinth. The papaverine hydrochloride diminishes the tonus of smooth muscle and consequently deals with any angiospasm in the vestibule. A course of treatment lasts six weeks, three monotrean tablets being taken daily after food. The course may have to be repeated two or three times. Of 23 patients (2 still under observation), 17 were completely cured and 2 considerably improved. The drug was ineffective in only 2 patients; 1 of these had an eighth nerve neuritis following overdosage with quinine for malaria and the other had a positive Wassermann reaction.

Biancalana [63] believes that the sympathetic fibers have an important part in vertigo. Partial or even complete section of the eighth nerve

62. von Bárány, A.: The Treatment of Otogenous Vertigo with Monotrean, Monatschr. f. Ohrenh. **73**:31 (Jan.) 1939.

63. Biancalana, L.: Resection of the Stellate Ganglion as a Treatment of Ménière's Syndrome, Ann. di laring., otol. **37**:41, 1939.

may be unsuccessful, and it then becomes necessary to interrupt the sympathetic fibers by resection of the stellate ganglion, which innervates a large part of the posterior cranial fossa through the vertebral nerve. Five patients are mentioned, of whom 4 underwent section of the eighth nerve but continued to have dizziness and tinnitus. All lost their dizziness after resection of the ganglion, and the tinnitus disappeared in some and became less in others. All showed the Claude Bernard-Horner syndrome after operation.

Three authors advise the injection of alcohol into the labyrinth in Ménière's disease. Berggren,[64] working through an aural speculum, incises the drum membrane inferiorly close to the annulus and folds the membrane upward, exposing the promontory. A small part of the promontory is denuded of mucosa, and a round burr on a long shank is driven by a dental handpiece to make a small hole into the promontory. When a dark spot comes into view, a needle is used to aspirate the endolymph and inject some drops of alcohol. The tympanic membrane is replaced and tamponed with iodoform-treated gauze.

The author does not mention the results except to state that he has operated only on patients with severe Ménière's disease who could not be kept free from attacks by prolonged treatment with phenobarbital and belladonna and whose hearing in the affected ear was poor.

Mollison[65] treated 50 patients with recurring attacks of vertigo by the injection of alcohol into the horizontal semicircular canal. The majority had so-called Ménière's disease, and some, but not all, had tried various medical treatments for the vertigo without relief. The procedure was done only when the hearing in the opposite ear was good, when the Wassermann test was negative and when the vertigo interfered seriously with normal activity and had continued over a period of one to nine years.

The technic of the operation consisted in opening of the mastoid process and antrum, with exposure of the horizontal semicircular canal. The latter was opened with a small gouge, and a few drops of dehydrated alcohol were then injected into the vestibule, after which the wound was sutured. Of the 50 patients, 74 per cent were cured, 10 per cent were unimproved and 16 per cent were only partly improved or were lost track of. One patient died some weeks after operation, from nephritis. Histologic examination showed infection in the mastoid cavity and diffuse suppurative labyrinthitis, presumably secondary to the mastoid infection.

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64. Berggren, E.: A Contribution Toward the Surgical Treatment of Morbus Ménière, Acta oto-laryng. **27**:626, 1939.

65. Mollison, W. W.: Surgical Treatment of Vertigo by Opening the External Semicircular Canal and Injecting Alcohol, Acta oto-laryng. **27**:222, 1939.

Cantele [66] reports a single case of Ménière's disease of four years' duration, in which the patient experienced no relief from medical treatment but obtained complete and permanent cure by the injection of alcohol into the horizontal semicircular canal.

[That surgical opening of the labyrinth with injection of alcohol is not entirely without risk is evidenced by Mollison's autopsy report of suppurative labyrinthitis following such treatment. The simple injection of alcohol through the oval window as described by Wright, Peacock and Yearsley in 1938 seems to involve less danger of suppurative labyrinthitis but some danger of injury to the facial nerve.]

Several authors advocate section of the eighth nerve or of the vestibular portion of the eighth nerve for Ménière's disease.

Berggren [67] reports 4 cases of Ménière's disease in which section of the vestibular nerve was performed by Olivecrona. Facial paralysis developed in 2. One patient recovered from his vertigo, but the tinnitus remained. The author stresses the importance of limiting surgical intervention to cases of genuine Ménière's disease with real invalidity and to those in which the treatment of Mygind and Dederding has been unsuccessful.

Olivecrona [68] states that Ménière's disease is best treated by operation if conservative treatment does not succeed in an adequate length of time. The operation is simple and not dangerous, and ten years' experience shows that there is no danger of recurrence of the vertigo, but the tinnitus remains even if the cochlear nerve is cut.

[The statement that the operation is "simple and not dangerous" is hardly compatible with the facial paralysis that developed in 2 of the 4 cases reported in the article by Berggren and with the deaths following section of the nerve which provided Hallpike with his first two specimens for histologic examination.]

Torkildsen [69] operated on a patient of 28 who had complained of tinnitus and vertigo since the age of 3. Examination showed deafness and decreased labyrinthine function. At operation the artery which runs with the facial and acoustic nerve into the internal acoustic meatus was found to be thicker than the facial nerve and pulsating. During the operation the patient had dizziness, which disappeared after the artery was ligated. After the operation there was no more dizziness. Tinnitus,

66. Cantele, P. G.: A Case of Ménière Syndrome Cured by Labyrinthotomy and Endolabyrinthine Alcohol Treatment, Valsalva **15**:326 (July) 1939.

67. Berggren, S.: Operative Treatment of Ménière's Syndrome, Acta otolaryng. **27**:202, 1939.

68. Olivecrona, H.: Ménière's Disease, Nord. med. (Hygiea) **1**:392 (Feb. 11) 1939.

69. Torkildsen, A.: Neurosurgical Treatment of Ménière's Disease, Nord. med. (Nord. med. tidskr.) **2**:1130 (April 15) 1939.

present the first day after operation, disappeared completely later on. The author suspects that the symptoms were due to congenital enlargement of the artery.

[Dandy has described 2 cases of Ménière's syndrome apparently due to an aberrant artery pressing on the eighth nerve.]

Shuster [70] presents an excellent review of vertigo with a discussion of causes, differentiation of types and interpretation of vestibular tests. He names the following etiologic factors:

1. General systemic conditions, such as cardiac disease, renal disease, arteriosclerosis and anemias (all due to disturbance of the blood supply to the labyrinth); air embolism; quinine and salicylates; tobacco; focal infection in the teeth, tonsils, sinuses and gallbladder, and dietetic errors.
2. Ocular muscle imbalance.
3. External otitis or impacted cerumen; occlusion of the eustachian tube, and acute and chronic suppurative otitis media.
4. Lesions of the brain in the frontal and parietal lobes and in the cerebellum.

In differentiation, a rotational vertigo is in general associated with aural lesions, with a vestibular type of nystagmus, deafness, nausea, vomiting and ataxia, with a tendency to rotate or fall in a direction opposite the quick component of the nystagmus. An irritable labyrinth causes deviation of the eyes toward the opposite side, with nystagmus toward the affected side and falling toward the opposite side.

Lesions of the posterior fossa give no sensitivity to tests and general depression of reactions. The lesion is usually on the side of depression or other abnormality. When both sides show abnormalities the lesion is usually in the midline.

Supratentorial lesions usually give marked sensitivity to tests and exaggerated responses. There is similar involvement on both sides with lesions of the midline. Interference with the vertical canal usually means that the lesion is on the opposite side. A perverted response of the horizontal canal to the caloric test means that the lesion is on the same side. Conjugate deviation is usually to the same side as the lesion.

The syndrome of a lesion of the cerebellopontile angle is recognized by loss of function on the affected side, including the hearing, and impairment of responses from the vertical canal and probably perversion of responses from the opposite horizontal canal.

An intracranial lesion is indicated by these spontaneous reactions: vertical nystagmus, horizontal nystagmus to one side, poor movements of the pelvic girdle.

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70. Shuster, B.: Vertigo: Clinical Consideration, Arch. Otolaryng. **30**:536 (Oct.) 1939.

An intracranial lesion is indicated by these vestibular reactions to tests: perverted nystagmus (oblique, vertical or horizontal instead of rotary); inverted nystagmus; large amplitude of nystagmus; small rapid ocular movements; dissociated ocular movements; conjugate deviation.

The treatment of vertigo requires, first, the differentiation of an intracranial from a peripheral lesion. In vertigo due to a peripheral lesion focal infection should be removed and dietetic irregularities and anemia or abnormal blood pressure corrected.

When no probable cause can be discovered, the intake of salt and fluid should be reduced and 6 capsules of 7½ grains (0.5 Gm.) of ammonium chloride should be taken three times a day for three days, then omitted for two days. Surgical section of the vestibular portion of the eighth nerve should be employed only as a last resort.

[Shuster has had an exceptionally large experience in the use of vestibular tests as an aid to diagnosis of intracranial conditions. His views are regarded by many as the most authoritative on this subject at the present time.]

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Abstracts from Current Literature

Ear

TREATMENT OF OTITIC MENINGITIS. D. S. CUNNING, Arch. Otolaryng. **30**:950 (Dec.) 1939.

According to Cunning, attempts at treatment of otitic meningitis have been disappointing. Reports covering several thousand cases indicate mortality rates of from 95 to 98 per cent. In this paper Cunning describes the treatment and results in 14 cases of otitic meningitis and in 1 case of meningitis following submucous resection. Only cases in which the pathogenic organism was actually recovered from the spinal fluid are included in this report. Treatment was similar in all cases, namely, a combination of radical surgical intervention, daily drainage of spinal fluid and intensive use of sulfanilamide or of one of the related compounds. Blood transfusions and intravenous administration of dextrose were employed during the same period. In this series of 15 consecutive cases of meningitis, 11 patients recovered and 4 died. Sulfanilamide, while obviously extremely important in treatment, is apparently not sufficient in itself to produce a cure. Surgical intervention in itself is not sufficient, and it seems that a combination of the two, namely, sulfanilamide and surgical intervention, offers the best hope. In the use of the drug it is important that treatment be started early, that the dose be large and that the treatment be continued for several weeks after the cessation of symptoms. Surgical measures, likewise, should be instituted as soon as the diagnosis is made. In 7 of 8 cases in which the invading organism was Streptococcus haemolyticus recovery occurred. In 2 cases in which the organism was pneumococcus type I and in 1 case in which the organism was pneumococcus type III death occurred. Had sulfapyridine been known when the pneumococcic infections were treated, the results might have been better. Six patients showed paralysis of the external rectus muscle. It seems that with the use of sulfanilamide plus surgical intervention meningitis is not necessarily to be considered as formidable as formerly.

J. A. M. A.

THE PROBLEM OF THE HARD OF HEARING SCHOOL CHILD. J. C. GEIGER, F. H. RODIN and B. A. CHASE, Arch. Pediat. **56**:605 (Sept.) 1939.

Over a period of ten years, three surveys were conducted on the hearing of the San Francisco school children. The survey was carried on in all the schools, and the hearing of children from grade 4 up to and including the high schools was tested. A 4-A audiometer was used in conducting the tests. When a borderline condition was encountered, the hearing was retested with a 2-A audiometer. The 2-A audiometer was also used to detect any change in the hearing after treatment and to follow up the progress of a defect in hearing. Over 86,000 children were examined.

A total of 36,191 children were examined during the first survey. On the retest, 3,427 had a loss of hearing of 9.0 or more per cent in the better ear—9.5 per cent of all the children under survey. The second survey, conducted five years later, included 30,713 children. On the retest 2,538 had a loss of 9.0 or more per cent in the better ear—8.3 per cent of all the children under survey. After a period of five years the third survey was conducted, and 19,627 children were examined. On the retest 1,433 children had a loss of 9.0 or more per cent in the better ear—7.3 per cent of the total number under survey. During the ten year period the number of children that had a loss of 9.0 or more per cent of their hearing dropped from 9.5 per cent to 7.3 per cent of all the children whose hearing was tested.

During the ten year period a marked reduction in the number of children with discharging ears occurred. In the first survey 0.5 per cent had discharging ears, while only 0.15 per cent had auricular manifestations in the last survey. In the first survey 355 children, or 1.0 per cent of all the children considered, had enlarged or infected tonsils associated with their defect in hearing. In the last survey 52 children, or 0.3 per cent of all children tested, showed abnormal tonsils with some hearing defect.

The reduction in the incidence of communicable diseases, with their many complications involving the ear, has reduced the incidence of loss of hearing. The early recognition of otitic complications and the prompt attention to such conditions have been important factors in reducing defects of hearing.

ORR, Buffalo. [AM. J. DIS. CHILD.]

THE MODE OF ACTION OF X-RAYS IN OTITIS MEDIA. L. L. TITCHE, New Orleans M. & S. J. **92**:203 (Oct.) 1939.

Titche reviews the literature pertaining to the action of roentgen rays on acute infections. Most especially, attention is drawn to the action on infections of the ear. The main action of roentgen rays seems to be to promote liquefaction and absorption of the inflammatory exudate and death of the organisms by destruction of leukocytes with liberation of lysin and antibodies.

The results in a series of 65 cases of ear infection are given. The series consisted of 35 cases of catarrhal otitis, 23 cases of acute purulent otitis and 7 cases of chronic purulent otitis.

The catarrhal patients were normal three days after a single irradiation with an average of 73.62 roentgens. Patients with acute purulent otitis received an average of 1.32 treatments with an average dose of 71.91 r. The ears were dry in nine days. Patients with chronic purulent otitis received an average of 1.33 treatments with an average of 75.38 r, and the ears were dry in 12.66 days.

The results, according to the author, speak for themselves. Any one may use roentgen treatment and expect results, but this form of therapy should not be relied on exclusively.

BERKLEY, Los Angeles. [AM. J. DIS. CHILD.]

SOME SCHOOL HEARING SURVEYS: ANALYSIS AND OBSERVATIONS. E. M. FREUND, New York State J. Med. **39**:1770 (Sept. 15) 1939.

A New York state law requires audiometric testing of all school children. Proper appliances and supervisors to carry out this law are provided. In 13,000 children 617 hearing defects have been found. Treatment, such as removal of tonsils and adenoids and attention to involved sinuses, should be carried out early if deafness is to be prevented. All children under 6 years of age found to have defects must be reported to the local health or welfare officer.

AIKMAN, Rochester, N. Y. [AM. J. DIS. CHILD.]

EVALUATION OF SULFANILAMIDE THERAPY IN ACUTE OTITIS MEDIA AND MASTOIDITIS. J. C. SCAL, New York State J. Med. **39**:1790 (Sept. 15) 1939.

Scal believes that sulfanilamide is contraindicated in the early stages of an ear infection. In acute mastoiditis, with sulfanilamide therapy the temperature may drop and all symptoms and tenderness disappear while roentgen examination still shows increasing destruction.

AIKMAN, Rochester, N. Y. [AM. J. DIS. CHILD.]

DIAGNOSIS AND TREATMENT OF INFANTILE MASTOIDITIS. P. W. LEATHART, Brit. M. J. **2**:723 (Oct. 7) 1939.

A discussion of mastoiditis occurring in infancy is outlined. The physical signs and symptoms and laboratory tests for the disease are mentioned. The point is

stressed that some infants succumb to rather evasive diseases which in fact have their origin in unrecognized mastoiditis. The condition is considered far more frequent by the author than has hitherto been believed. It is thought that early diagnosis can be made with confidence and that early operation can save a large percentage of these patients. The author states that babies should never be nursed or fed lying on their backs (this point is not made clear by him).

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

RECOVERY FROM OTOGENOUS STREPTOCOCCIC MENINGITIS FOLLOWING COMBINED SURGICAL AND SULFANILAMIDE TREATMENT. M. OMBRÉDANNE and A. PAPAIOANNOU, Arch. de méd. d. enf. **42**:376 (June) 1939.

A boy of 9½ years was seized with intense headache, nausea and vomiting, after a day on the beach. He had been exposed to the sun after bathing. The temperature rose to 39.9 C. (103.8 F.) and was maintained around this point for three days. He was admitted to a hospital for treatment. Examination gave negative results with the exception of slight coryza and congestion of the pharynx; the boy was badly sunburned. Within twenty-four hours, however, the temperature had reached 40 C. (104 F.). The headache was insupportable, and Kernig's sign was present. Spinal fluid obtained by puncture was very cloudy and contained 1,700 cells, mostly polymorphonuclears, but was free of bacteria on direct examination. Culture revealed a streptococcus, a finding which was confirmed by a further withdrawal of spinal fluid rich in streptococci. Treatment with sulfanilamide was begun on the first day. Up to this time there had been no complaint of pain in the auricular or mastoid regions, but on the following day latent otitis media was discovered on the right side. Paracentesis resulted in escape of pus containing streptococci. Under treatment with large doses of sulfanilamide, the meningeal symptoms rapidly subsided and the spinal fluid became sterile in six days. Mastoiditis now developed, with complete deafness of the right ear. A roentgen examination was confirmatory. Mastoidectomy showed considerable osseous destruction. The administration of sulfanilamide was continued for fourteen days. Facial paralysis was noted on the thirtenth day, and the patient was operated on again. Complete recovery followed.

AMESSE, Denver. [AM. J. DIS. CHILD.]

AURICULAR LESIONS FROM FULGURATION. A. C. REGHIZZI, Arch. ital. di otol. **52**:49 (Feb.) 1940.

In discussing the effects of electric discharges on the ear, Reghizzi refers to the reports of numerous writers and especially to the experiments on the ears of rabbits and guinea pigs which were conducted by Porta in 1932 and to the investigations by Masella of the chemical changes produced in the central and peripheral nervous system of rabbits by an alternating current of 150 volts and 50 cycles.

After the passage of an electric current through the ears of the animals, Porta observed that the effects were proportional to the strength, amount and duration of the current. Stronger currents caused fractures and other forms of trauma; with weaker currents of long duration, vasomotor and degenerative changes were more evident. Hemorrhages in the stria vascularis and the cochlea, slight degeneration of the sensorial elements and fracture of the stapes were observed after use of a strong current. In addition, there was degeneration of Deiters' nucleus, of the acoustic nerve and of the ganglions. With repeated applications of weak currents, vasomotor disturbances were less evident, but inflammatory reactions were pronounced.

Masella observed a temporary lowering of the calcium content of the nervous tissue and an increase of potassium and magnesium in the blood and the nervous system after application of the current. Cholesterol was increased in the blood and diminished in the peripheral nervous system.

Reghizzi observed a young man who was rendered unconscious by lightning. He was in a highly nervous state, complained of deafness, headache and vertigo and exhibited burns and characteristic cutaneous markings. Within twenty-four hours the right ear began to discharge. Examination revealed marked burns of the auricle and the canal, with much swelling, and perforation of the tympanic membrane. After one week the patient's condition was greatly improved, and he was well in two months except for slight deafness of the right ear.

Prognosis in such injuries is always uncertain. The capacity for harm from the electric current may vary quantitatively and qualitatively and may produce the most varied symptoms.

DENNIS, San Diego, Calif.

CHRONIC OTITIS MEDIA IN CHILDREN AS A GENERAL DISEASE WITH SPECIAL REFERENCE TO VITAMIN C METABOLISM. K. BERNFELD, M. FEILCHENFELD and W. HIRSCH, Ann. pædiat. **153**:222 (July) 1939.

Local treatment of chronic otitis media in children has not given favorable results. Prophylactic measures play a more important role. With a view to controlling the general factors influencing inflammations of the middle ear, 53 children from 6 months up to 10 years of age have been taken under clinical observation. A test for hypovitaminosis C was made by the saturation method in every child. Fifty per cent of these children had decreased resistance and were in poor nutritional condition. The values for hemoglobin were slightly below normal; the blood picture was normal. More than 30 per cent of the children showed tonsillar foci of infection and adenoid vegetations. In the majority suppurative otitis had lasted for one to three years. In more than 50 per cent of the patients with chronic otitis with the same nutrition a marked deficit in the output of vitamin C was found. The hypovitaminosis occurred in a higher percentage of the group of poorly nourished, slightly anemic children with decreased resistance than of the children in good condition. No relation could be detected between duration of illness and hypovitaminosis.

A series of children living under the same conditions but suffering from acute otitis were nearly all found to have a normal output of vitamin C. The hypovitaminosis C which is found in children with chronic otitis in contrast to those with acute otitis seems to relate to some constitutional factor.

FROM AUTHORS' SUMMARY. [AM. J. DIS. CHILD.]

Pharynx

TONSILLECTOMY IN THE DIABETIC CHILD. A. H. TERRY JR., New York State J. Med. **39**:1622 (Aug. 15) 1939.

Ether is the anesthetic of choice. The operation should be done in midday, as this provides plenty of time to give the proper diet. The usual amount of insulin is given in the morning. The amount required later must be determined. Fluid should be forced, but no drugs are given before operation. The operation should take as little time as possible.

AIKMAN, Rochester, N. Y. [AM. J. DIS. CHILD.]

THE PRESENT POSITION OF TONSIL OPERATIONS. MACLEOD YEARSLEY, Brit. J. Child. Dis. **36**:1 (Jan.-March) 1939.

"It would apear that large numbers of healthy tonsils are sacrificed annually, by over-zealous operation, in a slaughter of the innocents which out-Herods Herod."

A good part of the article consists of quotations from Alison Glover's paper on "The Incidence of Tonsillectomy in School Children." "If, as some believe,

the tonsils have a protective function . . . the child's entry to the environment of school might seem the time when they are most likely to be useful."

To remove tonsils is often a blunder, the real trouble being in the nose. The two points of importance in nasal physiologic function are a free airway and free drainage. In older children the nasal septum is often at fault, and a submucous resection may be done as early as the age of 12 years without interfering with proper nasal development.

Often removal of the adenoids alone will give beneficial results, especially in young children. The tonsils should be removed if they are definitely obstructing the fauces, if they are undoubtedly infected or if there is cervical adenitis. There are many more indications, but these are the most important.

LANGMANN, New York. [AM. J. DIS. CHILD.]

BENIGN ANGINA TONSILLARIS AND MYOCARDIAL DISEASE. G. W. PARADE, Med. Klin. **35**:269 (March) 1939.

The author points out on the basis of case histories of young adults that even mild tonsillar or pharyngeal infections may be complicated by myocardial damage in the form of conduction disturbances and disturbances in the electrocardiographic curve. There may be disturbance of the coronary circulation. Clinically the patient may have a slow convalescence and may suffer from dizziness, arrhythmia and anginal pain. The myocardial damage is apparently reversible, especially when the focus of infection is eliminated.

SIEMSEN, Chicago. [AM. J. DIS. CHILD.]

Larynx

ACUTE LARYNGOTRACHEOBRONCHITIS IN CHILDREN. CHEVALIER L. JACKSON, M. Clin. North America **22**:1518 (Nov.) 1938.

Although symptomatically it closely resembles laryngotracheobronchial diphtheria, this disease is not diphtheria. Of Jackson's cases, the condition in 85 per cent was unquestionably due to streptococci. Other organisms (staphylococci and influenza bacilli) have been reported as predominant in some cases. The clinical picture is that of a more or less toxic child with a moderate or high temperature and rapidly progressive obstructive dyspnea. Absence of membrane in the oropharynx does not absolutely preclude the presence of diphtheria, and when visualization of the lower air passages is not possible it is advisable to give an injection of serum. Serious complications, such as infecton of the blood stream (especially with streptococcic conditions), subglottic edema and bronchopneumonia may occur. Obstructive atelectasis is frequently mistaken for pneumonic consolidation. Atelectatic areas quickly disappear, however, when bronchial obstruction is removed.

Until recently the mortality of the fulminating types was 50 to 70 per cent; with the employment of all modern methods of treatment, the death rate is seldom less than 25 per cent.

The two essentials of treatment are water and a free airway. Fluids must be supplied in adequate amounts by whatever means available. The child should be kept in an environment of warm moist air and given every possible chance to rest. However, sedatives, especially opiates, should be avoided. The restless child will become quiet if his dyspnea is relieved. For the relief of obstruction, tracheotomy should be done as promptly as indicated, and the passages should be kept clear by means of suction through the tube. This will produce relief in about 50 per cent of cases. Later it may be necessary to remove hard masses plugging the bronchi, through the bronchoscope. In some cases this may have to be done repeatedly as a life-saving measure.

EUGENE H. SMITH, Ogden, Utah. [AM. J. DIS. CHILD.]

FATAL FIBRINOUS LARYNGITIS AND TRACHEITIS. FRITIOF LEEGAARD, Nord. med. tidskr. **16**:1566 (Oct. 1) 1938.

An 8 year old boy fell ill after a slight common cold with signs of laryngeal stenosis, which in the beginning was diagnosed as pseudocroup. He had fever (temperature 40.2 C. [104.3 F.]). There was no cyanosis and no pharyngitis. The mucous membrane of the subglottic trachea was swollen. During the night there was a sudden increase in symptoms of stenosis followed by loss of consciousness. Tracheotomy was performed, without success. At postmortem examination membranous laryngotracheitis was demonstrated. Hemolytic staphylococci were found. There was no diphtheria. Death was considered due to cardiac paralysis caused by the septic condition.

WALLGREN, Göteborg, Sweden, [AM. J. DIS. CHILD.]

Miscellaneous

THE TREATMENT OF PNEUMOCOCCIC MENINGITIS WITH SULFANILAMIDE: REVIEW OF THE LITERATURE AND REPORT OF SIX ADDITIONAL CASES. BARBARA A. HEWELL and A. GRAEME MITCHELL, J. A. M. A. **112**:1033 (March 18) 1939.

The authors report that before sulfanilamide was used all of a series of 23 patients with pneumococcic meningitis died. Since the use of sulfanilamide or of related compounds was begun, at least 30 recoveries are known, and the authors add 3 from their 6 cases in which the drug was used. The results show a definite beneficial effect of this type of chemotherapy.

HEERSMA, Kalamazoo, Mich. [AM. J. DIS. CHILD.]

ALLERGY OF THE UPPER RESPIRATORY TRACT IN INFANCY AND CHILDHOOD. G. PINESS and H. MILLER, J. A. M. A. **113**:734 (Aug. 26) 1939.

Comparing the incidence of attempts at surgical cure of allergic conditions at the present time with that in the past, the authors deplore the fact that 43 per cent of asthmatic patients were advised to have tonsillectomies to relieve their asthma. They and others find the promises of relief unfulfilled.

Part of the difficulty is caused by confusing the symptoms of allergy with those of infection and those of the combination of the two conditions. The authors urge physicians to recognize the signs of allergy and not to be swept away into a preference for tonsillectomy, blinded to the actual cause of disease of the upper respiratory tract.

HEERSMA, Kalamazoo, Mich. [AM. J. DIS. CHILD.]

THE ROLE OF ALLERGY IN BRONCHIECTASIS. SAMUEL H. WATSON and CHARLES S. KIBLER, J. Allergy **10**:364 (May) 1939.

A study of 46 cases of bronchiectasis in the clinic of the authors at Tucson, Ariz., revealed that a high percentage of patients had definite evidence of allergy (hay fever, asthma, nasal polyps, atopic dermatitis and abnormal amounts of eosinophils in the nasal or bronchial secretions) exclusive of positive cutaneous tests. They feel that bronchiectasis can be closely simulated by basal allergic bronchitis, that bronchitis can be favorably influenced by allergic management and that the association of sinusitis and bronchiectasis can be explained on a basis of allergy.

HOYER, Cincinnati. [AM. J. DIS. CHILD.]

BRONCHOGRAPHY IN CHILDREN. H. E. S. PEARSON and H. L. THORNTON, Brit. J. Radiol. **12**:229 (April) 1939.

A method of introducing iodized poppyseed oil into the bronchi of small children is described. After basal anesthesia has been induced by rectal administration of paraldehyde 5 per cent cocaine is sprayed into the pharynx and nose. Nitrogen monoxide and oxygen anesthesia is then induced, and a large intratracheal tube in

which a small indwelling catheter lies is placed in the trachea. The anesthetic is administered through the large tube, and the oil is instilled through the catheter, which enters the large tube through a small side opening near the top of the tube. With the child under nitrogen monoxide anesthesia, the oil is then injected into the trachea, and its flow is watched and directed by fluoroscopic observation. Any part of the lung can be examined by inverting or turning the child. Films are taken as desired.

SQUIRE, Chicago. [AM. J. DIS. CHILD.]

MODERN CONCEPTIONS OF ALLERGY IN CHILDREN. A. E. LARGUIA, Día méd. **11**:431 (May 22) 1939; Medicos **1**:17 (Nov.) 1939.

Larguia discusses modern conceptions of the development, character and types of allergy in children. He uses the word "allergy" as a synonym of "hypersensitivity." The term covers all the specific allergic reactions which cannot be considered in the group of anaphylactic phenomena. According to the author, heredity is the factor of primary importance in the development of allergy. From 40 to 60 per cent of the children who suffer from allergy come from families with a history of allergy, whereas only 5 per cent of nonallergic children come from such families. Children with a bilateral history of familial allergy have a great predisposition to the development of allergic diseases. The larger the number of cases of inheritance in the family the more marked the influence of heredity and the earlier the development of allergy in the children (Cook, Spain). Hyperallergic children frequently show reactions to the allergens to which the parents react (Woringer). The frequency of hereditary allergic transmission is twice as great in females as in males. It seems that the majority of allergic children belong to only one blood group. Hereditary transmission of allergy follows Mendel's laws. It is of a recessive character and either of a conceptional or germinal type, but the transmission may be congenital (transplacental) or may appear after birth or during early infancy. The conception of an intrauterine sensitization of a constitutionally predisposed fetus by proteins which pass through the placenta explains the early appearance of allergic eczema or asthma in the infant who is fed on the mother's breast alone. A larger number of children (more than 60 per cent) who suffer from allergic diseases give positive skin reactions to egg albumin and other antigens with which they have had no contact. A great number of such positive skin reactions, however, are due to potential sensitivity of the patients for the given antigens, as the patients have, at one and the same time, actual and potential sensitization. The allergic reaction depends on a constitutional predisposition of certain structures, namely, the skin and the nasal, bronchial and conjunctival mucosae, the digestive tract and possibly, also, the brain, the retina and the bladder, to experience a shock when they come in contact with a specific allergen. During the shock specific antibodies are produced which, in association with the allergens and in the presence of certain substances in the blood, cause the allergic symptoms. Heredity plays no part in the development of acquired allergy. In the course of acquired allergy, which is milder than hereditary allergy, the skin reactions are frequently negative or induced by larger doses of allergens, and frequently the antibodies cannot be identified in the blood of the patients. The diagnosis of acquired allergy is difficult. The treatment consists in eliminating contact with the given allergen. Certain hyperallergic patients who suffer from hereditary allergy may also suffer from acquired allergy.

FROM THE AUTHOR'S SUMMARY. [AM. J. DIS. CHILD.]

CAUSATION AND FREQUENCY OF RETROBULBAR NEURITIS. P. CIBIS, Klin. Monatsbl. f. Augenh. **102**:205 (Feb.) 1939.

In a paper well illustrated with tables, Cibis renders an account of the etiologic factors in 189 cases of retrobulbar neuritis observed at the ophthalmic clinic of the University of Heidelberg, in Germany, during the last eighteen years. The findings are discussed and compared with those at other ophthalmic hospitals.

Acute retrobulbar neuritis was present in 133 cases, multiple sclerosis prevailing in 56 of these cases, while the etiologic factor remained unknown in 55. In the remaining 22 cases, sinusitis, acute poliomyelitis, grip, infection of the teeth and other acute infections were present. Multiple sclerosis was suspected in 22 of the 55 cases in which the etiologic agent was not established. After elimination of all those cases in which some other etiologic agent was found, including arteriosclerosis and amblyopia caused by tobacco and alcohol, multiple sclerosis was found to be the cause of retrobulbar neuritis in 70 per cent of the 189 cases. This figure tallies with that of Fleischer (66 per cent) and of Langenbeck (77 per cent). The ages of Cibis' patients ranged between 14 and 78. One of the tables demonstrates that the frequency of retrobulbar neuritis did not increase from year to year correlative with improvement in diagnosis but varied irregularly. Cibis stresses the predominating frequency of multiple sclerosis as an etiologic factor in retrobulbar neuritis. The frequency of multiple sclerosis observed by Cibis paralleled the frequency of this disease recorded for the same years at the neurologic hospital at Heidelberg.

STOLL, Cincinnati. [ARCH. OPHTH.]

THE DEAF-MUTE: A MEDICOSOCIAL STUDY. R. LABBÉ, Rev. méd.-sociale et de protec. de l'enf. **7**:1 (Jan.-Feb.); 88 (March-April); 161 (May-June); 258 (July-Aug.) 1939.

In the opinion of the author, who is chief physician of the National Institute for Deaf-Mutes in Paris, the average deaf-mute is born normal except for his auditory defect. This defect, although purely physical, brings about from the beginning a retardation of mental development which it is difficult to combat. (All degrees of native intelligence are found among deaf-mutes.) The influence of heredity, although obscure, and of family environment is more evident among deaf-mutes than among other persons.

Through education and training given the deaf-mutes in special institutions, particularly through demutization, most deaf-mutes may become able to earn their living.

The author discusses in detail the method of instruction and demutization, vocational education, mental and character training and the historical development of schools for deaf-mutes in France. A list of such schools in France is given.

BIERMAN, Washington, D. C. [AM. J. DIS. CHILD.]

Society Transactions

AMERICAN OTOLOGICAL SOCIETY

HORACE NEWHART, M.D., *President*

ABSTRACTED BY ALFRED LEWY, M.D.

Seventy-Third Annual Meeting, Rye, N. Y., May 30-31, 1940

President's Address: Observations on the Conservation of Hearing. DR. HORACE NEWHART, Minneapolis.

Achievement in the prevention and alleviation of poor hearing until recently has been slow. There have been extenuating circumstances: the anatomic inaccessibility of the ear; the inadequacy of classic hearing tests; the widespread belief that efforts to prevent, cure or compensate are futile; the indifference to organized efforts at conservation, and the lack among physicians and educators of familiarity with fundamental facts pertaining to the subject.

Research has disclosed many previously unrecognized causes of impairment of hearing, and clinical experience shows that many patients for whom treatment has hitherto been hopeless can be helped.

The most effective means for reducing the high incidence of hearing defects is the periodic testing of school children by audiometers. It is predicted that audiometric tests will be made routinely of registrants in educational institutions and of members of industrial, military, naval and various civic groups. Already they are finding application in aviation and in the health programs of industrial organizations.

The use of the audiometer in the public schools is not a step toward socialized medicine. It increases the work of the private practitioner by disclosing unsuspected hearing defects. The use of the phonograph audiometer has provided a more accurate and economic screening test than other methods. In testing children too young to write, monitors must be used, to whom the numbers heard are whispered. The technic may have to be revised to include pure tones of high pitch, say 11,548 cycles.

The use of standard audiogram blanks and uniform recording symbols is desirable. There should be a revision of terminology. For lesser degrees than total, "hard of hearing," "hearing deficient," "or hearing defect" or "impairment" should be preferred.

The committee on nomenclature of the Conference of Executives of Schools for the Deaf has adopted the following:

1. (*a*) "congenitally deaf"
   (*b*) "adventitiously deaf" (deafness acquired)
2. "hard of hearing," hearing defective but functional with or without a hearing aid.

It is recommended that the American Otological Society appoint a committee on nomenclature. A research of special importance would be the investigation of industrial deafness, its evidence, causes and prevention.

Recognition, Treatment and Prevention of Hearing Impairment in Children. DR. S. J. CROWE, Baltimore.

(This paper is a summary of a Baltimore group study and concerns an investigation of 700 children, who must be followed up for five to ten years for final conclusions.)

For the removal or reduction of lymphoid tissue in and around the eustachian tube, which cannot be removed surgically, radium appears the safest and best agent.

During the past sixteen years, audiometer, fork and voice tests have been made on 15,000 patients, approximately 3,000 of whom were children. The audiometer range was 32 to 16384 double vibrations. To prevent deafness, studies must be concentrated on children. If adenoids are removed before puberty, they recur in 75 per cent of the patients and interfere with the ventilation of the eustachian tube. Lymphoid tissue is an integral part of the mucosa in this area, and complete removal is impossible.

The same types of middle and internal ear deafness occur in children as in adults, but study and treatment are more successful in children, in whom these changes are just beginning. Partial obstruction of the eustachian tube for a period of years causes, first, hyperemia of the middle ear, then the formation of secretory, then of myxomatous and finally of fibrous tissue. Otoscopically, the first changes are seen in Shrapnell's membrane. Patency of the eustachian tube is determined by retraction, especially in Shrapnell's membrane, examination with the nasopharyngoscope, voice test with the opposite ear masked and the audiometer with 8000 double vibrations and up.

Regardless of the primary cause, every child with obstruction of the eustachian tube should be treated. No atrophic changes follow the proper dosage of radon. This should not be given during acute nasopharyngitis. Radiation is of no value in the treatment for suppuration, otosclerosis or inner ear deafness. To be of value, it must be applied before the middle and inner ear are irreparably damaged.

Irradiation of Hypertrophic Lymphoid Tissue. Dr. Curtis F. Burnam, Baltimore.

Irradiation of hyertrophic lymphoid tissue by roentgen rays is not as safe as that by radium but includes the entire eustachian tube. Most of the obstructive lymphoid tissue is in the nasopharynx; therefore, radon placed in the nasopharynx is safer. Roentgen rays should be delivered through portals of not over 4 cm. square surface. Radon in a glass container requires thirty days to reach equilibrium. The applicator can be passed along the floor of the nose and taped so that the glass container is not in contact with the nasal mucosa. The whole applicator is 13 cm. long, the glass container 1 to 1.5 cm., in a brass tube with a lumen of 1.5 mm. and walls 1 mm. thick. The gamma rays constitute 95.5 per cent and the beta 4.5 per cent. The hard beta rays have 8 or 9 mm. penetration. The dose in roentgens (r) figures about 500. The erythema dose is 1,400 r. Two gram minutes is the average therapeutic dose. From 600 to 659 r may be given if both sides are irradiated at one sitting. The necessity for repetition depends on the results of hearing tests.

Histologic Studies of the Eustachian Tubes of Persons with Good Hearing. Dr. Leroy Polvogt and Dr. Dudley C. Babb, Baltimore.

There is marked variation in the histologic structure of the eustachian tube in persons with good hearing. The tunica propria is thick in the pharyngeal portion but thins out toward the middle ear. The number of glands varies greatly, as does the vascularity, which is usually greater in children. The ciliated epithelium frequently extends to the middle ear. Goblet cells vary greatly in number. The tube may be straight and smooth but usually looks corrugated from evaginations of the tunica propria. The evaginations contain blood vessels and sometimes lymphoid nodules. The tube may run through solid bone or highly pneumatized bone, and there may be tubal cells lined with ciliated epithelium continuous with the main part of the tube. These may form a pathway for infection and are a potential danger in bougienage. The most important findings are the lymphoid nodules, which may be deep in the tunica propria or near the surface and are found even in late adult life. The part in deafness which is played by lymphoid tissue has not yet been studied histologically.

Impaired Hearing in School Children. Dr. Stacy R. Guild, Dr. Leroy M. Polvogt, Dr. Harold R. Sandstead, Dr. Walter E. Loch, Dr. Ella Langer, Dr. Mary H. Robbins and Dr. W. Andrew Parr, Baltimore.

The report is based on examinations of 1,365 school children between the ages of 8 and 14, carried out during the school year 1939-1940. All examinations were made at the hospital and by physicians. The thresholds for 14 tones from 32 to 16384 double vibrations were determined in a "sound-proof" room with a Western Electric 1-A audiometer, with masking when necessary. Weber, Schwabach and Rinne tests were made with a fork of 512 double vibrations. The nasopharynx was examined with a nasopharyngoscope. Treatment was advised for 239 of the children. Two hundred and eight are having radon treatment of the nasopharynx. One hundred and eighty-four have had from one to four treatments over a period of two to six months. Reexamination of these shows remarkable shrinkage of lymphoid tissue, and in 95 the adenoids are definitely smaller. Eustachian orifices formerly overgrown are now free. Four general types of average audiogram are shown: (1) approximately normal (allowing a dip at one frequency, usually 4096); (2) good hearing up to 8192, then abrupt decline; (3) abrupt decline around 4096; (4) a gradual decline beginning about 2048. Four and three tenths per cent of the children, whose audiograms are not included in these four types, had impairment for all tones. The high tone impairment occurring alone would not have been discovered by the phonograph group and speech method. Until many children with loss for high tones only have been observed over a period of years, the clinical significance of this type of impairment cannot be properly evaluated, but it is apparent that a better means than the phonograph audiometer should be devised to discover children in need of preventive otologic care.

Of interest are the findings of bilateral good hearing in 70 per cent of the girls and in only 47.3 per cent of the boys, of greater incidence of deafness in winter than in autumn and of the fact that more boys than girls had had tonsils and adenoids removed.

No influence of the condition of the teeth or of underweight was demonstrated, nor was any relationship found to anthropologic measurements.

The appearance of the tympanic membrane was not a good index of hearing. Forty-four and five tenths per cent of children with marked retraction showed no hearing loss, and good hearing was found in many children with lymphoid tissue overgrowing the eustachian orifice. However, this is considered a menace requiring attention.

The radon therapy was effective in reducing the lymphoid hypertrophy and improving hearing for high tones. More time for another check-up is needed, to evaluate recurrences and the influence on hearing loss due to other causes and particularly to determine the prognostic value of the discovery of hearing loss for high tones.

Two important facts are a challenge to the profession. Over 40 per cent of all children examined do not hear all tones within the range of intensity regarded as normal, and 75 per cent of children attending school have nasopharyngeal conditions that are a potential menace to hearing.

DISCUSSION OF PAPERS OF DR. CROWE; DR. BURNAM; DR. POLVOGT AND DR. BABB, AND DR. GUILD AND ASSOCIATES

Dr. Edmund Prince Fowler, New York: There has been presented a treatment by radon therapy for lymphoid tissue in the vicinity of or covering the tubal aperture. This treatment is logical if such tissue interferes with the function of the middle ear. Some years ago I had in mind accomplishing this by means of a eustachian bougie carrying radium. The procedure was difficult to carry out on children and I never knew whether the results were from radium, the bougienage or the treatment that went with it.

A dependable test for proper functioning of the tube is needed. The Toynbee test (movement of the membrane during swallowing) is good if there are no adhesions. In administering hearing tests a few months apart, one must remember

that the practice factor may give the impression that hearing is better. Dr. Crowe's children have pretty good hearing; nevertheless, if the loss for high tones is a precursor of deafness, the slightly lower tones should also be important in predicting progressive deafness.

There can be no clearcut frequency above which a loss means potential deafness and below which it does not mean this. Children with slight degrees of deafness also usually show this high tone loss. (Illustrative slides were shown.)

His children (224 with binaural, 40 with monaural impairment), with 10 to 20 decibel loss, were divided into four groups: (*a*) those with high frequency loss, (*b*) those with equal frequency loss (horizontal), (*c*) those with middle or speech frequency loss (256 to 4096) and (*d*) those with low frequency loss. They were also classified into age groups. In the groups with slight and moderate loss, less than 16 per cent showed high frequency loss predominating. Only in the group in which there was severe deafness were high tone losses impressive, 50 per cent. There was no preponderance of low tone loss in any group. In my experience, hearing losses above 8000 cycles also include the 8000 cycle tone. The high tones vary in normal ears. The average for high tones is not known. At 12000 cycles, even at 20 to 25 decibels over threshold, very close attention is required, even in a sound-proof room, and a child may easily give misleading answers.

There were 321 children with monaural impairment with 10 to 20 decibel loss, showing a level type of deafness, so that in at least 585 children, with the exception of the severely binaurally deaf, the percentage of greatest loss in the high frequencies is very small and would have been of less value for early diagnosis than would such an estimate for the middle or low tones.

Dr. George M. Coates, Philadelphia: This symposium of Dr. Crowe and his associates is of the greatest interest to every member of this society, and all are indebted to them for bringing up this matter of lymphoid hyperplasia, which was close to the heart of Dr. D. B. Delavan, who requested a study of this subject by the American Laryngological Association. The question has not been answered but will have to be. The method advocated by Dr. Crowe is treatment, and if the formation of lymphoid tissue can be prevented, even more will have been accomplished. Dr. Crowe stated that after 75 per cent of operations for adenoids there are recurrences. This is probably true. The operation should be checked by finger exploration. It is my opinion that it is inadequately done in the majority of cases. Rosenmüller's fossa often escapes attention, and lymphoid tissue in the lumen of the tube cannot be reached surgically.

Osteopaths for years have practiced "bloodless" surgical treatment of the tube. Sometimes the improvement is remarkable, but it is usually temporary, because the hyperplasia recurs or scar tissue is formed. To obtain the greatest benefit, the loss of hearing must be detected early, and if the early loss is in high tones the methods of examination must be revised.

Does this mean that audiometers must be reequipped?

One can determine whether the lymphoid tissue is in the fossa of Rosenmüller and around the orifice of the tube only by means of the nasopharyngoscope. In examining a group of children, pick out a few steady ones who will not object. This will make easier the handling of the rest. The examination is not painful and can be done on the average child.

For applying 1000 millicuries of radon, only two minutes are necessary. Roentgen treatment is not satisfactory in my hands. Perhaps the results claimed for the Stokes roentgen treatment were due to the effect on the lymphoid tissue.

The treatment should be in the hands of experts in otorhinology and should be supervised by trained radiologists. I am not without some apprehension that the method, if the latter becomes popular, may lead to disastrous results.

Why children with conductive deafness should show earliest loss in high tones, I do not understand.

The study of the causes of the lymphoid hypertrophy will have to be continued and an uninterrupted search made for early impairment of hearing.

Better methods of examining school children will have to be developed.

DR. MAX A. GOLDSTEIN, St. Louis: Most audiometers do not include frequencies above 10000 cycles. Therefore, few otologists have had the opportunity of observing the upper tone ranges in normality or defect. This is rather a new phase of the determination of hearing ranges, but it may be an index to beginning deterioration of hearing in children. It is another form of approach to this important object of study. I believe the Johns Hopkins group have proved that they are getting some results with radon. In this country there are but few places where a radon tube of the type used by Dr. Burnam can be prepared, and the expense is prohibitive. Do I understand that the usual dose is 1,200 millicuries applied for two minutes? Is there an advantage in using radon rather than radium, which requires forty minutes' irradiation as a minimum dose? Is it true that there are few places where there are 5 Gm. of radium to be had?

DR. CURTIS F. BURNAM, Baltimore: My associates and I have been using radon because it was available in concentrations that permitted the treatment of a large number of children with the same applicator in a short time. The children do not mind the applicator, once it is in place. Some object to the introduction. There is no difference in the effects of radon and radium. The cost of radium is about $20 a milligram, so an applicator can be bought for approximately $1,000, and in about an hour's treatment a patient can be given the required dose. A modern x-ray machine costs $12,000 or $13,000, and certainly $2,000 or $3,000 will furnish as many applicators as any one or any ordinary clinic would desire.

The technic of the application of radium salts is exactly like that of the application of radon, i. e., the salts are introduced through the nostril into the nasopharynx and left to give the desired dose. The filtration may not be ideal and the intervals of application may be improved. My associates and I attempt to treat in a way that won't harm.

DR. J. C. STEINBERG, New York: Many records of hearing tests were collected at the World's Fair. For every instance of medium and low tone loss of 25 decibels, there were 4 of high tone deafness. For every 100 boys aged 10 to 19 having this high tone loss, there were 46 girls similarly affected.

In seven different surveys, covering thousands of children in various parts of the country, the average was 68 girls to 100 boys (2,300 in Missouri were equally divided). This suggests the importance of a close study of these defects. The incidence per hundred varied from 3 to 13, according to the physician examining. The average incidence was 6 per cent.

DR. STACY R. GUILD, Baltimore: The incidence reported is very different when a nasopharyngoscope is used.

DR. MOSES H. LURIE, Boston: Have you any data on the relation of high tone loss to otitis media, with or without scarlet fever, measles and mumps?

DR. STACY R. GUILD, Baltimore: Scarlet fever and measles caused only a small part of the total deafness. Deafness from mumps is usually so severe as to require special classes. Most of the children with 9 per cent or more loss as tested by the phonograph audiometer had a history of otitis media and changes in the tympanic membrane.

DR. HAROLD R. SANDSTEAD, Baltimore: A negative history as to a previous attack of otitis is unreliable. Many children with changes in the tympanic membrane had good hearing.

DR. THOMAS C. GALLOWAY, Evanston, Ill.: Emphasis has been put on hearing defects as a mechanical problem. May there not be subacute or chronic infection acting through absorption of toxin? This might explain loss of high tones in accordance with the previous conception. Perhaps radiation acts by elimination of toxins or by control of infections, local or otherwise. Has study been given to the more specific infections which otologists know may be associated with perception deafness?

DR. WALTER E. LOCH, Baltimore: Medical students and children with normal and with impaired hearing were tested. The external canal was occluded without pressure on the tympanic membrane. The occlusion caused loss of hearing for

middle and high tones and only a slight loss for low tones. The experiments prove that conductive hearing impairment may cause marked loss in the high tone region. Careful inquiry was made as to previous infectious disease.

Dr. James A. Babbitt, Philadelphia: (Dr. Babbitt reemphasized Dr. Crowe's remarks on the need of precision in the surgical care of lymphoid hyperplasia.) The question of tonsils is less important.

Dr. Henry M. Goodyear, Cincinnati: Has any study been made of the reduction of lymphoid tissue by medication, e. g., with iodine, iron, thyroid and parathyroid? When considering operation, one must not overlook hypertrophies which occur with the eruption of the molar teeth, excessive intake of sugar, causing swelling and watery discharge, and excessive intake of starch, causing tendency toward heavy mucous secretion. How does Dr. Crowe feel about supervising operation by direct vision of the nasopharynx?

Dr. S. J. Crowe, Baltimore: I do not approve of retracting the soft palate, because of the temporary postoperative paralysis. I prefer the use of the nasopharyngoscope.

Dr. Burt P. Shurly, Detroit: Tonsillectomies and adenoidectomies constitute 50 per cent of all operations in the country and involve a great deal of the time and financial support of the profession. The adenoid operation is often badly performed by the general practitioner and the general surgeon. Examinations of school children will bring out just where the deficiencies of hearing begin.

In the Detroit schools there have been 100,000 examinations by technicians. It is the duty of the examiner to refer those requiring treatment to the family physician. Dr. Crowe's method is more scientific. The WPA will assist any properly sponsored organization to carry on examinations of school children, but only three or four states have taken advantage of this. I think the question of national examination should have general interest. If it is learned some day that the lymphoid ring is influenced by vitamins B and A, otolaryngologists will be glad to have the findings concerning that come before them.

Dr. S. J. Crowe, Baltimore: The questions imply interest, not criticism. The interest in the prevention and alleviation of poor hearing is much greater than it was ten or fifteen years ago.

Clarification of Certain Phases of the Physiology of Hearing. Dr. Hallowell Davis, Boston.

The intra-aural muscles contract in response to loud sounds (of about 50 decibels or more). They are a protection against overstimulation. In facial paralysis, loud sounds in the affected ear become annoying.

Bone conduction acts on the inner ear by (1) inertia of the ossicular chain and (2) compression of the bony labyrinth. The second mechanism is dominant if the ossicular chain is absent, ankylosed or otherwise restrained. If the ear is too sensitive to bone conduction, the internal head noises, such as blood movement, chewing and voice, form a background which masks external sounds. The ossicular chain is so evenly balanced that its center of gravity is not more than 0.33 mm. from its axis of rotation, and if its function is impeded the sound of a tuning fork is lateralized to the affected side.

The sensation of pitch is determined by the position of the vibratory pattern on the basilar membrane and not by the frequency of impulses in the auditory nerve. The optimal positions for various frequencies have been plotted by Culler, by means of electric microphones, and confirmed by others.

The location of abrupt high tone deafness in the basal turn had already been determined by Crowe, Guild and Polvogt. Another approach, through psychophysical measurements, has led to almost identical maps. The shift necessary to produce a just noticeable difference corresponds approximately to the distance occupied by two internal hair cells (Stevens and Volkmann, corroborated by Fletcher, Davis and Lurie).

A map of the basilar membrane may be constructed which is consistent with these six sets of data:

1. Position of maximum electrical activity.
2. Impairment of electrical activity by localized lesions.
3. Pathologic changes associated with high tone deafness.
4. Integration of just noticeable difference of pitch.
5. Contribution of different parts of the basilar membrane to the total loudness of sound.
6. The relation of pitch subjectively perceived to frequency.

Frequency 2200 lies at the midpoint of the basilar membrane. The low octaves are more and more compressed as the apex is approached. With this map Fletcher has calculated the auditory patterns of steamboat whistles, street noises, bugle notes and other sounds.

If a carefully adjusted alternating current is passed through the head (the zone between hearing, tickle, pain and vertigo is very narrow), a musical note may be heard one octave above the current frequency, as the membrane is attracted to the promontory twice for each cycle, or if the membrane is missing and the current is applied to the round window, a tone corresponding to the current frequency is heard. This electrophone effect is the inverse of the usual aural microphonic effect. When the fibers of a particular part of the basilar membrane are excited, a tone is heard; if the fibers are selected at random by the current, a noise is heard. The analyzing mechanism of the inner ear remains an essential part of the mechanism of hearing. Incidentally, Stevens finds the frequency theory untenable.

DISCUSSION

DR. WALTER HUGHSON, Abington, Pa.: Dr. Davis directed attention first to the intrinsic musculature. The intra-aural muscles contract on stimulation of the external canal at 50 or 60 decibels, low tones, and become tactile. According to Luscher, in facial paralysis the level of the lesion can be diagnosed by the effect on the stapedius muscle. Experimental data indicate a protective function of the intrinsic musculature. It is possible for middle ear lesions to impair bone conduction to an extent equal to the impairment by neural lesions. Unexplained impairments of bone conduction may be due to the contour or the density of the skull. Some patients prefer to wear the bone conduction receiver on the maxilla or zygoma. The ear can be too sensitive to bone conduction when this is not masked by air conduction.

Experimentally, changes in intracochlear pressure have a profound effect on hearing. This has been demonstrated clinically in the drainage of spinal fluid. Dr. Guild has referred to seasonal recurrence of hearing impairment in children. Adults are also affected by temperature and humidity, both as to auditory and as to vestibular symptoms, especially tinnitus. This must be remembered in appraising any therapeutic effort.

Why do all lesions, toxic, vascular or mechanical, affect high frequencies first? When an alternating current is passed through the head 30 decibels below air conduction, how does fatigue develop? Only noise and vertigo are experienced. It hardly seems that this mass stimulation has any application to the frequency theory.

DR. M. H. LURIE, Boston: I should like to mention the Pohlman apparatus, composed of an artificial drum and a bristle, a replica of the bird's drum and single ossicle. This apparatus transmits air waves to either the region of the oval or that of the round window. By testing, one can find the exact spot where the fluid waves can be set in motion. This apparatus is useful in cases in which membrane and ossicles are absent.

When the tympanic membrane is absent, electric stimulation of the cochlea is done by a beat oscillator connected to the round or to the oval window. In patients with lesions of the middle ear, with drums present, there is no response to

any stimulus short of pain-producing current. In patients who had had radical mastoidectomies, with the electrode at the oval window one volt caused pain and vertigo, sometimes nystagmus and even facial stimulation. Two patients in whom the cochlea had been exenterated gave no response. Patients who had had ossiculectomy or radical mastoidectomy could recognize pitch and intensity; some had islands of deafness and some a complete range of hearing. The difference between a tone and noise could be recognized. Too much voltage caused a labyrinthine reaction. Patients without a membrane but with ossicles had normal range. I believe (1) that the cochlear fluid was put into motion and (2) that the auditory nerve was stimulated. When the nerve was directly stimulated, the result was noise plus vestibular disturbance.

DR. EDMUND P. FOWLER, New York: What is the effect of applying bone conduction equally from both sides? One may have a means for differentiating a squeeze on one side and an impedance on the other.

HARVEY FLETCHER, PH.D., New York: A sharp percussion sound has more of the sound energy in the high tones; therefore, assuming that all the nerve is equally susceptible, the greatest damaging acoustic shock is in the high tones. The area where the high frequencies are sensed is nearest the outside, where the sound wave reaches first. These sounds are not pure tone but agitate the entire length of the basilar membrane.

STACY R. GUILD, Ph.D., Baltimore: There is no reason from clinical tests and sections to change the statement that from 1000 double vibrations down there is no localization in the human cochlea.

DR. HALLOWELL DAVIS, Boston: I want to thank the discussers, especially Drs. Hughson and Lurie, for additional information. It has been observed that the external hair cells in animals are more susceptible of damage. They are exposed to greater mechanical movement. There is an exception, however. Degeneration may be pretty well localized to the neighborhood of a loud tone.

Electrophonic effect has some bearing. According to the frequency theory, one should expect that 60 or 100 cycles electrically would result in a tone of similar frequency, but often merely noise is heard. Perhaps the frequency theory does not hold for low tones, or perhaps it needs further elaboration. Dr. Fowler assures me he has not read Bárány's monograph, so he must have arrived independently at the same conclusion. He differentiated between a pushing from side to side and simultaneous compression. If simultaneous stimulation reenforces, it must be a compression wave. If it were a side to side wave, a simultaneous push on both sides would tend to cancel.

Clinical Objectives in the Chemotherapy of Hemolytic Streptococcus Infections. DR. CHAMP LYONS and DR. ROBERT NORTON GANZ, Boston.

The protective antibody against hemolytic streptococcus is antibacterial. Since there is no commercial supply of immune serum, the immunotransfusion was used to advantage before sulfanilamide became available. It was later demonstrated that sulfanilamide and the bacterial antibody were synergistic.

Studies were made on the importance of the effect of antibacterial antibody on the course of an infection against which sulfanilamide treatment is being used. In the cases studied there were no foci of undrained pus. It appears that delay of cure and recurrences of infection occur predominantly in patients who lack antibacterial antibody.

The administration of sulfanilamide for nonsuppurating infection leads to significant clinical improvement in twelve hours and apparent arrest in forty-eight hours. Patients with no antibacterial antibody may fail to respond to chemotherapy. When toxic symptoms develop in such patients, immunotransfusion is advised, and one may then change to sulfapyridine.

Persistent fever may be observed in patients possessing antibacterial antibody and receiving sulfanilamide. It may be noted in (1) patients with scarlet fever, (2) those with undrained pus or necrotic tissue, (3) those with endocarditis, throm-

bophlebitis or periphlebitis, especially intracranial, and (4) those with drug fever. If a skin rash is not present, the condition is difficult to diagnose. If antibody is present, one may stop administering the drug and observe.

Immunotransfusion is more reliable than transfusion of merely compatible blood, and if no anemia is present serum alone may be used.

Failure of chemotherapy demands investigation of the following possible causes:

1. Inadequate blood concentrations of the chemotherapeutic agent.
2. Deficiency of antibacterial antibody.
3. Coexistent erythrogenic toxemia of scarlet fever.
4. Undrained pus or necrotic tissue.
5. Intravascular or perivascular sepsis, either thrombophlebitis or endocarditis.
6. Drug toxemia.

DISCUSSION

Dr. Harry P. Schenck, Philadelphia: The balance between recovery and death may depend on immunity.

The infections may be divided into three groups:

1. Diffuse, unlocalized infection, responding to drug therapy alone.
2. Localized, necrotizing infection, requiring therapy and surgical drainage.
3. An intermediate group for which drug and serum therapy suffice.

Many of Dr. Lyons' patients had infections in group 3, except those who showed drug sensitivity. Specific immunity should be increased by immunotransfusion, vaccination or active immunization. Vaccines and lysates have been more useful in pneumococcus infection.

Meningitis due to Pneumococcus type XVIII suddenly developed in a nurse after she had apparently recovered from an acute infection of the upper respiratory tract. The left sphenoid sinus was drained surgically, and with sulfanilamide therapy prompt recovery ensued. A month later there was a recurrence. A small sequestrum was removed and sulfapyridine given, with recovery. Two months later there was another recurrence, with Pneumococcus type XVIII; sulfathiazole and a lysate were given. Surgical intervention was not indicated. Again there was prompt recovery. There has been no recurrence in three months, and the pulse rate, which remained rapid throughout, is now normal. This illustrates the value of conferring immunity after apparent cure.

When there is destruction of mastoid cells, surgical drainage is imperative.

Dr. William E. Grove, Milwaukee: Did the outcome result from chemotherapy alone, the immune serum alone or from a synergistic action? One cannot experiment with separate groups of human beings. Reactions have occurred in compatible cases under chemotherapy. Serum may be used instead of whole blood when there is no anemia to be treated. The masking of danger signals by chemotherapy is known to all otologists.

Dr. Charles F. Porter, Boston: Three years ago I was severely criticized for predicting that the laboratory would aid progress in the management of bone disease. Dr. Lyons has today proved that it has. Does early use of sulfanilamide or sulfapyridine preclude the formation of antibodies by the patient?

Dr. Ralph A. Fenton, Portland, Ore.: I should like to have Dr. Lyons' comment on the number of patients who have had chemotherapy alone, with masking of symptoms. Also, has Dr. Lyons used Osgood's marrow culture to determine synergistic values? Osgood has reported synergistic effects of sulfathiazole with arsphenamine in cardiac infections due to Streptococcus viridans.

Dr. Thomas C. Galloway, Evanston, Ill.: Will Dr. Lyons give us his idea on withholding chemotherapy until immune bodies have been developed? Does he agree with Mellon that fastness may develop, as in the case of antispirochetal drugs? Does the presence of peptone in necrotic tissue preserve bacteria? How may heparin act in vascular disease?

DR. CHAMP LYONS, Boston: Vaccines and lysates have their role in combating pneumococcus infections. Active immunization is not very successful against hemolytic streptococcus.

Slow withdrawal of chemotherapy may be useful, but there may be toxic reactions from prolonged administration. I have tried heavy dosage to conclude treatment in seven to ten days.

Infections of bone are more resistant than are those of soft parts, but chemotherapy may permit primary closure after sequestrotomy necessitated by metastatic infarction following sinus thrombophlebitis.

The formation of antibodies does not appear to be retarded by chemotherapy. I favor early use of sulfanilamide, supplemented by such serums as may be necessary.

I have not used marrow cultures. It has been shown that the combination of chemotherapy and antibacterial antibody kills more bacteria in human blood than either alone.

With a combination of neoarsphenamine and sulfathiazole I have no experience. I have yet to see an unquestionable viridans, hemolytic streptococcic or pneumococcic endocarditis cured. Staphylococcic endocarditis may heal. I have not seen "fastness" to sulfanilamide in the hemolytic streptococcus. "Fastness" may be observed with sulfapyridine treatment againt Pneumococcus.

Bacterial survival within an abscess is probably due to the inability of leukocytes to function within this zone rather than to the presence of peptone. Heparin decreases coagulability. It will not dissolve an established thrombus but will prevent further collection of platelets and thrombus and so prevent the building up of an aseptic thrombosis. I am still experimenting with it on surgically inaccessible septic thrombophlebitis. I am still observing the effects of topical application. Skin may be grafted over infected areas by using sulfathiazole. The drug opens a new approach to local dressings.

The General Problem of Deafness in the Population. DR. WILLIS C. BEASLEY, Bethesda, Md.

This report contains data collected by the United States Public Health Service during the national health survey (1935-1936) and the clinical investigation of hearing supplementing the household census. Consideration is given to the prevalence of deafness in relation to age, sex, income, employment status and occupation, and to the prevalence of deafness for high tones in relation to age and sex. Attention is given to the loss of hearing for high tones in childhood and to chronic progressive deafness in adults. No relation has as yet been demonstrated. For purposes of survey, the country was divided into geographic areas, and from each of these samples of the population in thirty-one cities of over 100,000 population were taken. House to house canvasses were made. In 70 per cent of the cases the information was obtained from members of the family who were not deaf. Seventeen clinics were set up in twelve cities. Both deaf and nondeaf members of families were invited to enter the clinics for study. A good audiometric index of ability to hear speech sounds was believed to be obtained by averaging hearing loss for 1024 and 2048 cycles. The subjects were divided into five groups according to the stage of deafness:

1. Persons who hear speech at close range but have difficulty in group conversation and in hearing in public places (audiometeric loss, 47 decibels on 1024 and 2048 cycles).
2. Persons who hear on the telephone or hear loud speech (55 decibel loss).
3. Persons who hear only with artificial aids (68 decibel loss).
4. Persons with total deafness for speech, acquired after learning speech, (89 decibel loss).
5. Congenitally deaf persons and those who acquired deafness before speech was learned.

The observation is made that in all stages of deafness there is usually loss by bone conduction; therefore, impairments are predominantly nerve deafness.

Persons in the first stage of deafness (group 1) according to the social history (household census) averaged a 47 decibel loss. According to the clinical history, a group in the same stage averaged a 24 decibel loss, showing by the household census that many cases are not reported.

In the age group 5 to 14 years the household census showed an incidence of deafness of 2.95 per thousand for boys and 2.26 for girls. Except in the age group 25 to 34, deafness is more prevalent among males. According to age, the incidence rises gradually from 2.95 per thousand for males in the 5 to 14 year group to 175.08 at the age of 75 and over; for females it rises from 2.26 to 135.95 per thousand.

An estimate of new cases is attempted. The prevalence of deafness among persons with an income of $3,000 per annum is 100 per hundred thousand; among those with an income of $1,000 or less, it is 174 to 190, according to age, in males and 138 to 224 in females. This is attributed partly to greater exposure in males and to their tendency to fall into lower income groups because of the disability. The subjective borderline for noticeable defects is between 20 and 25 decibels. A range of 750 to 3,000 cycles is all that is necessary for recognition of speech sounds. Lower and higher frequencies influence the quality of sound perceived. There is no evidence that losses of 10,000 cycles and above in childhood have any relation to losses of 3,500 to 8,000 in adults or are demonstrably associated with nerve lesions or precursors of greater loss in the next decade.

The article (*Laryngoscope* **50**:856 [Sept.] 1940) is replete with statistical graphs and tables, from which the foregoing and other inferences are drawn. There is a considerable discrepancy in the percentage of deafness found in those applying at clinics and those reported by the household census. This is attributed to the fact that in 70 per cent of the cases the census information was obtained from other members of the family, from whom the deafness had been concealed.

DISCUSSION

DR. BURT R. SHURLY, Detroit: The early diagnosis of deafness is highly important. By early diagnosis of tuberculosis, the death rate from that disease has been moved from first to seventh place. The investigation reported was a tremendous and worthy undertaking, and so is the research work at the Johns Hopkins University, bringing in another field, but these patients, with few exceptions, have not been referred for treatment. In the survey of the Detroit Board of Education, it was shown that among 267,000 children 4.6 per cent had defective hearing. These were given cards to their family physicians. A family may choose its own physician or specialist, and the trained nurse and the teacher follow this up.

When the census taker comes around, 77 per cent of the answers to his questions are given by women of the household, perhaps by the hired girl. The percentage of error in the information must be high.

The public health services of the various states can do a splendid job under proper medical direction.

(A series of slides was shown to illustrate the Detroit system of making auditory surveys and the examination of army men.)

DR. RALPH A. FENTON, Portland, Ore.: The result of this survey is comparable with the information obtained at the time of the World War, but that information was not analyzed and correlated to nutritional deficiency, occupation, exposure, toxic habits (tobacco and alcohol) and particularly to economic status.

In Oregon the Children's Bureau, the Committee for Prevention of Deafness, the state medical society, the state board of health and the superintendent of public instruction have cooperated. The children requiring treatment have been referred for that purpose, and physicians have regained control of children they had lost

sight of. Unless the local medical group is called into cooperation, projects such as these do not succeed. The United States Public Health Service always went to the local medical society first.

This statistical material brings out the axiom, long maintained by the American Medical Association, that improved health is not dependent so much on completely sponsored medical relief as on the economic level of the population. These statistics show that the highest incidence of deafness was among the poor.

Dr. Edmund Prince Fowler, New York: The amount of unemployment among intellectual workers differs markedly from that in the toiler group, so it is impossible to define a single standard based solely on hearing loss. Only a court can decide the "percentage of disability" that would apply in different cases.

Many surveys have been made. The making of surveys should be stopped and something done about the ones already made.

This year a bill was presented to the New York legislature. It failed because some department disliked some item in it. It will go through next year. During the last war I saw in my division dozens of men with running ears. This defect in the mass causes little incapacity for duty.

Dr. Max A. Goldstein, St. Louis: What is the economic value of statistics? There are complete case records, in triplicate, of 50,000 children in St. Louis County. These children were followed up, and those needing lip reading were so instructed. They are no longer "repeaters" in the class room.

I think the problem should be summarized as an otologic responsibility for cooperation with the school authorities. If all recommendations are not consistently followed up, they must be of lessened value in the prevention of deafness.

A New Amplifier Having Characteristics Similar to Those of the Human Ear and Its Application to the Problem of Deafness. F. H. Shepard Jr., B.S., M.E., Merchantville, N. J.

This paper deals with: (1) the nonlinear characteristics of the normal human ear for various levels of sound intensity and for various frequencies; (2) with the manner in which these amplitude frequency characteristics enable the normal ear to handle tremendous ranges of volume; (3) the manner in which they enable the normal listener to hear intelligibly at various distances from the source of sound and under varied acoustic conditions, and (4) the way in which these same characteristics, effectively incorporated in a hearing aid amplifier, will without apparent distortion fill in frequency losses in a person's audiogram and will increase his range of hearing even in the presence of strong nearby masking sounds.

DISCUSSION

Harvey Fletcher, Ph.D., New York: Fifteen years ago I published an account of the phenomenon described. If several frequencies, say 300, 400, 500 and 600, are put into a circuit, they are heard as a 100 cycle tone. I do not think, however, that this contributes to the improvement of a hearing aid. In severe deafness, the range that can be heard between pain at the top level and the threshold at the bottom is narrow. The thing to do is to compress the range of intensity. The circuit described in this paper does this. My associates and I made a set like that for Mr. Edison, amplifying the high frequencies and using a compressor, namely, a nonlinear amplifier of the type suggested. He was able to hear very well with this set but did not wear it because he considered the inconvenience more than the improvement in hearing was worth.

As to the theory: When one is listening to speech and goes away from the speaker, the high tones are lost first, because of the absorption in the room, as stated, but there are other factors. The high frequencies go out in a beam, like light, and the low ones spread everywhere. If there were no absorption, the low tones would be lost more rapidly than the high, so one could not tell in a room

which of the two frequencies was predominant. I doubt if an amplifier could be made to compensate for that fact and operate in different types of rooms. If one had a tone gap, a nonuniform response on a nonlinear amplifier would spread somewhat over the gap. I can't see that spreading over the gap would be a help.

Amplifiers of this sort are no doubt helpful in severe types of deafness, and I think more of them should be used.

DR. HALLOWELL DAVIS, Boston: The loudness of sound and the electrical output of the cochlea are not necessarily the same as assumed by Mr. Shepard. The two probably do not follow the same curve, but the general argument is perfectly good.

A nonlinear increase in the manner in which the nervous system adds up nerve impulses will not necessarily introduce subjective harmonics. The mechanics of the cochlea will. I think the operation of Mr. Shepard's apparatus should be regarded as an empiric proposition. If it works—more power to it. The demonstration was convincing.

MR. F. H. SHEPARD JR., Merchantville, N. J.: If I had known where Dr. Fletcher had described the phenomenon of tone recognition by its harmonics, I should have referred to his work. I intended no claim to such discovery. My contribution is a simple amplifier that generates the proper harmonics within itself. The variable amounts of distortion at various frequencies can be controlled at will. As Dr. Fletcher has pointed out, no device can be made to compensate completely for differences in timbre in different rooms. The human ear does not have this facility; however, its characteristics do facilitate intelligibility under varied conditions and do keep the apparent timbre of sounds heard at varied distances more nearly constant. As I tried to explain in the paper, a hearing aid should copy the compromise of nature as exemplified in the normal human ear.

Perhaps I did not stress strongly enough the fact that one of the principal features of this type of amplifier is its ability to handle with equal facility a whisper, a pistol shot or distant sounds in the presence of nearby masking sounds. When the compression or distortion of this type amplifier is properly set, the listener is not conscious of any distortion or unnaturalness of the sounds heard.

PANEL DISCUSSIONS

L. W. DEAN, M.D., St. Louis, *Presiding*

INFECTIOUS THROMBOPHLEBITIS OF THE LATERAL SINUS

The audience will assume that every statement made by a member of the panel is approved by the others unless challenged. In the latter event a clear statement of each opininon shall be made.

1. Conditions Simulating Sinus Thrombophlebitis.

DR. ALEXIS F. HARTMANN, St. Louis: Otologists should first decide what drug to use in chemotherapy. Sulfanilamide affects chiefly the hemolytic streptococcus, which is the causative agent in 80 to 90 per cent of the instances of infectious thrombophlebitis. From 15 to 20 per cent of patients may not be affected by this drug.

I should administer sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) or sulfathiazole (2-sulfanilamidothiazole) to a blood concentration of 5 to 10 mg. per hundred cubic centimeters, which means 0.2 Gm. per kilogram, or 1.5 grains (0.09 Gm.) per pound per day, and I should give one half of this in the initial dose. If any doubt arose as to retention, I should give the drug parenterally. Sulfanilamide may be given in 0.5 or 1 per cent solution intravenously or subcutaneously. Sulfapyridine and sulfathiazole may be given in a solution of 5 per cent; the sodium salt, intravenously or, in a more dilute solution, subcutaneously. After it

has been established that one is dealing with a streptococcus, one may switch to sulfanilamide. Other important needs must be met, such as dehydration and chemical changes, ketosis, acidosis, occasionally alkalosis and, in infections of longer duration, loss of red cells and plasma. Fluids of the proper sort may be used to combat these conditions.

DR. FREDERICK M. LAW, New York: Sulfanilamide produces an increased transparency in the roentgen film, which may cause error unless a film of the normal mastoid is available for comparison. Perhaps there is a change in the mucosa which causes this increased transparency. This change takes but a few days.

DR. A. C. FURSTENBERG, Ann Arbor, Mich.: For six months I have used sulfanilamide, 10 or 15 Gm. administered locally, after mastoid operation. I close the upper two thirds of the incision and insert a small pack in the independent portion. I believe the ear dries sooner with this treatment, but there is a tendency for the mastoid to bleed. The cavity fills with clot, and the drug tends to keep it sterile. Some of these wounds have healed, and the patients have been discharged with the wounds closed in ten to eleven days after operation. I have had similar results in osteomyelitis of the skull, in spite of the claim that in most instances this disease is caused by a staphylococcic infection. The wounds have remained admirably clean. The surgeons have used the drug in suppurations of the neck and mediastinum. As much as 20 Gm. have been used in an osteomyelitic defect of the skull, with no ill effects.

DR. HAROLD I. LILLIE, Rochester, Minn.: It seems to me that I have seen reports of interference with the normal healing of wounds.

DR. A. C. FURSTENBERG, Ann Arbor, Mich.: This experiment is still in the early stage. The wound discharges have been mostly serous, sometimes profuse but not purulent.

DR. ALEXIS F. HARTMANN, St. Louis: The reported cases were those of clean stomach wounds, and healing was judged by pulling the wounds apart. The reports of many orthopedic surgeons are favorable for the control of infection in compound fractures by the use of the drug.

DR. HAROLD I. LILLIE, Rochester, Minn.: What about bleeding from soft tissues?

DR. A. C. FURSTENBERG, Ann Arbor, Mich.: Yes, there is a tendency toward increased bleeding from soft tissues.

DR. HAROLD I. LILLIE, Rochester, Minn.: Sulfamethylthiazole (2-sulfanilamido-4-methylthiazole) appeared to us at the Mayo Clinic to be a good hemostatic for wounds of the frontal sinus and mastoid. The house officer even used it for the control of post-tonsillar bleeding. This drug has been recalled by the manufacturers because it supposedly causes spinoneural paralysis. (ED. NOTE.—Because of its tendency to cause undesirable reactions this drug has never been licensed for sale in the United States.)

DR. FREDERICK M. LAW, New York: If the discharge is thinner, it would make the change I suggested on the film.

DR. ALEXIS F. HARTMANN, St. Louis: Ten milligrams per hundred cubic centimeters in the blood would hardly affect the specific gravity or the transmissibility by roentgen rays.

It was found experimentally that the effect of sulfanilamide was proportional to its concentration and inverse to the number of organisms present. It had no direct action—there was a lag of several hours until sulfanilamide produced some active substance. The most widely held theory is that of Mayer and Shaffer—that partial oxidation takes place yielding bactericidal products. In the absence of a good supply of oxygen, these are not present. Dr. Furstenberg's observations indicate the contrary. In Chicago I cited a case of irrigation of a septic knee joint, which

became sterile before mastoidectomy was performed. I think the keeping of mastoid wounds clean by irrigation with 1 per cent sulfanilamide in physiologic solution of sodium chloride is of help.

DR. A. C. FURSTENBERG, Ann Arbor, Mich.: I think I should use sulfanilamide for Streptococcus and sulfapyridine for Pneumococcus.

DR. HAROLD I. LILLIE, Rochester, Minn.: I think these drugs mask symptoms of intracranial complications as they do those of acute mastoiditis. Their use should be reserved for the complications of mastoiditis and not for the initial infection, else otologists must learn to interpret a new set of signs. I have had this experience, and I have observed the recurrence of symptoms of meningitis on discontinuance of the drug after using it over what seemed to be a sufficient period. It is necessary to know what may recur after the patient is dismissed, apparently well.

DR. L. W. DEAN, St. Louis: If the patient is receiving chemotherapy and you cannct diagnose the complication, do you stop the drug and watch the course of the disease?

DR. HAROLD I. LILLIE, Rochester, Minn.: Yes. It has been a difficult problem.

DR. ALEXIS F. HARTMANN, St. Louis: I do not feel that one can tell in any case whether an infection will stay mild or will get worse. I think the most good is done by energetically treating all potentially serious infections in the nose and throat with sulfanilamide or one of the derivatives of this drug.

DR. FREDERICK M. LAW, New York: If sulfanilamide is given after osseous change has started, roentgen examination will show a continuance of the destructive process. If no change has taken place, the symptoms are relieved and the cell structure remains unchanged. Whatever change there is remains stationary while the drug is taken; when this is stopped, the destructive process goes on. It is best to use follow-up films. Stereoscopic films will show cells located under the cortex, which remain intact the longest.

DR. ISIDORE FRIESNER, New York: Has it not been shown that the concentration of these drugs in the body tissues parallels that in the blood except in bone and fat, where it is considerably less? I do not believe these drugs influence infection in the bone. I do not understand how their local application to the bone can influence the process. Probably the healing tissues, the granulomas that are well vascularized, are amenable. If there is a discharge, there is an undrained focus in the bone. I have yet to see clinical proof that sulfanilamide influences a process in the bone. In my experience, most cases of mastoiditis do not give a picture of extension from the middle ear but from the beginning give evidence of bone involvement. One may be called an abscess of the middle ear, the other a panosteitis of the temporal bone from the beginning. The latter condition is not influenced by sulfanilamide. When would you use this drug in acute otitis media?

DR. ALEXIS F. HARTMANN, St. Louis: There is a difference when the drug is administered early in the course of the disease and when it is given late. Experimental observations on mice have shown that if peritonitis progressed too far no concentration of the drug sterilized the peritoneal cavity. Bone marrow was sterilized by 1 mg. if the drug was administered shortly after the introduction of infection, but no concentration was effective after eighteen hours.

DR. A. C. FURSTENBERG, Ann Arbor, Mich.: The drug has little effect on extensive infections of the calvarium, but all otologists know its effect on infection in soft tissues—as erysipelas, pneumonia and meningitis. I see no contraindications to its use at any time in infections of the upper respiratory tract. The drug infrequently has harmful results. One can observe toxic effects in cutaneous manifestations and stop administration. In the presence of reduction of hemoglobin and of severe gastrointestinal symptoms and when the drug is suspected of causing fever, administration can be stopped a day or two for observation. Discontinue the drug when you think the patient has recovered.

DR. ISIDORE FRIESNER, New York: I did not make myself clear. When the ear appears to have healed, the drug is discontinued, but the symptoms recur. How can you judge the patient's condition without stopping the drug for observation for possible postauricular disease?

DR. L. W. DEAN, St. Louis: Can you tell what is in the sinus after septic thrombophlebitis has apparently healed or in the apparently healed mastoid?

DR. ALEXIS F. HARTMANN, St. Louis: Thrombophlebitis may be followed by complete healing of the drumhead. I try for at most three weeks to eradicate this infection by chemotherapy; I then drain the mastoid but continue the chemotherapy to prevent postoperative complications. One has to do almost as many operations with chemotherapy as without it unless one sees the patients very early in the course of the disease. This year we have had more mastoid operations than for a long time, despite chemotherapy.

DR. ISIDORE FRIESNER, New York: I should like to obviate the complications that occur if I fail to make the diagnosis of suppurative disease in the bone. Chemotherapy destroys the physical signs on which I have learned to depend. The evidence but not the disease is destroyed; even the roentgenogram is affected.

DR. FREDERICK M. LAW, New York: I have not had a patient with coalescent mastoid recover under sulfanilamide therapy without operation. I did observe such recoveries before I used sulfanilamide, and the mastoids eventually returned to the normal state, according to the films.

If the roentgenogram shows a lack of bony septums, the condition is potentially dangerous. Base judgment on the roentgenogram.

DR. ISIDORE FRIESNER, New York: I pay considerable attention to the roentgen evidence in continued suppuration.

DR. HAROLD I. LILLIE, Rochester, Minn.: I agree with Dr. Friesner that a case should be managed from the onset without the drug so that symptoms of impending complications can be recognized. Sulfanilamide is sold to lay people for all kinds of conditions. Much harm may result from its indiscriminate use.

DR. ISIDORE FRIESNER, New York: I think the drug should be given early and then the administration stopped for observations. It will not cure mastoid disease, and despite its use intracranial complications will occur.

DR. ALEXIS F. HARTMANN, St. Louis: I should not give just a little of the drug for a few days and then stop, lest I be fooled by appearances. I should give full doses at the beginning.

DR. A. C. FURSTENBERG, Ann Arbor, Mich.: I have little apprehension about the average otologist's postponing operation too long. I agree with Dr. Hartmann: sulfanilamide is a drug that tends to prevent serious complications. Why discontinue using it? But at the same time, one should not fail to recognize mastoid cases in which surgical intervention is required.

DR. ISIDORE FRIESNER, New York: How are you going to make a diagnosis in a case in which the disease in an ear has resolved with good hearing when there is no change in the canal wall and the mastoid is full of pus? Latent infections such as this occur much more frequently now, and I wouldn't depend 100 per cent on roentgenograms.

DR. L. W. DEAN, St. Louis: Has any member seen masking of intracranial complications by the drug?

DR. ISIDORE FRIESNER, New York: I have seen a patient with sympathetic meningitis from an epidural abscess, who had no aural discharge and gave evidence of resolution of the pathologic process in the middle ear. The roentgenogram was reported negative. I believe sulfanilamide was a contributing factor.

2. Papilledema: Its Etiology, Clinical Significance and Treatment.

DR. ISIDORE FRIESNER, New York: There must be several factors responsible, because not all the patients had increased intracranial pressure and not all had an obturating thrombus. In ten years my associates and I encountered 25 cases of papilledema. All the patients had restoration of normal vision, and the mortality was 4 per cent. Some had metastatic lesions, some not; some had prolonged bacteremia after operation. There appeared to be no connection between the severity of the disease and the papilledema. In some patients who had an obturating thrombus, the papilledema increased after ligation. Papilledema appears to be due not to venous embarrassment, intracranial pressure or toxicity alone but probably to some combination that I am not clear about.

DR. L. W. DEAN, St. Louis: In twenty-five years of careful observation of the fundi in cases of accidental opening of the sinus and packing to stop hemorrhage, my associates and I have not seen evidence of papilledema with an uninfected sinus.

DR. A. C. FURSTENBERG, Ann Arbor: It has been reported that a small jugular vein on one side with a thrombus of the sigmoid sinus on the other has caused papilledema because of disturbed intracranial circulation. I have had no experience with such an anomaly. One may not encounter a condition such as this in a lifetime.

DR. HAROLD I. LILLIE, Rochester, Minn.: The papilledema I have observed came on after operation, in contradistinction to phlebitis. I interpreted it as caused by an influence on the petrosal sinus. The probabilities are that papilledema is due to a combination of conditions, as stated by Dr. Friesner.

DR. ISIDORE FRIESNER, New York: In 12 cases of our series, the papilledema appeared before operation and in 13, after; in 1 it appeared twenty-eight days, in 1 thirty odd days and in 1 forty-one days after operation. There was no evidence of increased intracranial pressure.

DR. ALEXIS F. HARTMANN, St. Louis: Papilledema was present in 14 of 34 cases in which it was looked for and was doubtful in 4 cases. All the patients with papilledema had a positive Queckenstedt test, but not all in whom the Queckenstedt test was positive had papilledema. All in whom a bilateral Queckenstedt was positive died. Optic atrophy eventually developed in 1 patient.

3. Intracranial Complications: Their Diagnosis and Treatment.

DR. HAROLD I. LILLIE, Rochester, Minn.: I had a case of sinus phlebitis in which cerebellar abscess developed.

DR. HARRIS P. MOSHER, Boston: My associates and I must have had 5 or 6.

DR. A. C. FURSTENBERG, Ann Arbor, Mich.: I believe brain abscesses occur from neighborhood infections, by continuity of tissue. We investigated a series of cases and demonstrated that in 78 per cent the infection occurred by perivascular extension along a stalk. It is rare for the visceral wall of the sinus to rupture and permit the formation of a stalk.

DR. ISIDORE FRIESNER, New York: From the literature it appears that brain abscesses from sinus thrombosis occur almost invariably in the posterior fossa. I encountered a temporosphenoidal abscess. Most of the deaths in the cases encountered by me were from meningitis.

DR. L. W. DEAN, St. Louis: In forty years only 2 abscesses, both cerebellar, secondary to sinus thrombosis were diagnosed on my service. In 1 case there was a hole in the inner surface of the sinus.

DR. ALEXIS F. HARTMANN, St. Louis: (A slide was shown.) This child was admitted to the hospital in a somnolent condition; spinal fluid was turbid; there was an enormous loss of mastoid substance; the sinus was unrecognizable; there was a parasinoidal abscess; the dura was necrotic, and when it was uncovered, out gushed an ounce (31 cc.) of foul pus. There was no sign of abscess.

DR. HAROLD I. LILLIE, Rochester, Minn.: There is a subdural abscess by direct extension and a deeper abscess in a lobule from infarction or perivascular infiltration. The subdural abscess is a brain suppuration adjacent to the focus of infection and carries a higher mortality.

4. Diagnosis of Anatomic Variations of the Sinus.

DR. A. C. FURSTENBERG, Ann Arbor, Mich.: In over 4,000 cadavers I have found 9 anomalies of the intracranial sinuses. I will relate my own clinical experience and theorize on procedures for others. (Slides were shown.)

In slide 1 the transverse or sigmoid portion is double. One might easily overlook a thrombus in one only.

Slide 2 shows that the transverse portion may be absent and a large superior petrosal sinus may continue into the sigmoid. In this case control of hemorrhage was difficult and had to be accomplished by packing introduced into the lumen of the large superior petrosal sinus.

Slide 3 shows an enlarged emissary vein; in the presence of this condition, special effort may be required for ablation or control of hemorrhage.

In slide 4 the sigmoid sinus ends in a blind pouch below; the flow is through an enormous emissary vein or a large petrosquamosal sinus into the external jugular vein (as in mammals below the anthropoid apes). The large emissary vein and the petrosquamosal sinus must be dealt with.

Slide 5 shows that the lateral sinus may be in the scalp.

Slide 6 shows that the transverse and sigmoid portions may contain lakes, with severe hemorrhage when opened.

In slide 7 a large petrosquamosal sinus carries blood from the lateral sinus to the soft tissues in front of and below the temporal region.

DR. FREDERICK M. LAW, New York: The roentgenogram would not pick up these anomalies.

DR. HAROLD I. LILLIE, Rochester, Minn.: I once found a bifurcation of the internal jugular vein.

DR. ISIDORE FRIESNER, New York: I have seen lakes in a sigmoid sinus several times.

DR. FREDERICK M. LAW, New York: The roentgenogram shows the heavy groove, not the clot, and cannot be depended on for a diagnosis of sinus thrombosis or as an indication of the size of the jugular foramen. Erosion of the sinus plate may sometimes be diagnosed if one has previous films for comparison.

DR. A. C. FURSTENBERG, Ann Arbor, Mich.: I think the size of the groove indicates the size of the membranous lateral sinus, and frequently one can observe that the right is larger than the left.

DR. FREDERICK M. LAW, New York: The sharply defined anterior border can be seen, and it is assumed that the termination of the emissary vein is at the posterior border, which gives some idea of size. The appearance of position depends on the technic used in making the roentgenogram and on the depth of the grooves. All one can say is that the sinus is forward or back.

PANEL DISCUSSION

FREDERICK T. HILL, M.D., Waterville, Maine, *Chairman*

Information Please: Regarding Hearing Aids and Audiometers.

MR. HOWARD A. CARTER, Chicago, representing the Council of Physical Therapy of the American Medical Association: In 1936 the Council on Physical Therapy appointed a group of consultants. The first audiometer was accepted in 1940; the first hearing aid, in 1935. Members of the Council and consultants are not paid.

About six hundred pages of communications have been put out. A report of the work of the consultants will soon be published in *The Journal of the American Medical Association.*

These hard working consultants are: Dr. Bunch, Dr. Coates, Dr. Fowler, Dr. Hayden (now deceased), Dr. Jones, Dr. MacFarlan, Dr. Newhart, Dr. Shurly, Dr. Wherry, Dr. Nash, and Dr. Paul Sabin, of Riverbank Laboratories. Dr. Beasley has aided to some extent.

When application is made for approval of either an audiometer or a hearing aid, the firm which manufactures the appliance is furnished copies of the official rules governing advertising and related matters and of the minimum requirements for acceptance of a product. Advertising statements considered questionable by the Council or by the consultants are taken up with the firm, which ordinarily cooperates and withdraws them. The Jones-Knudsen audiometer was accepted a year ago but is not on the market. The other two accepted are the Maico and the Western Electric 6-B. The preparation of minimum requirements takes a long time and much work, and the manufacturers have had difficulty in meeting these requirements.

DR. ARTHUR W. PROETZ, St. Louis: For clinical use, I prefer an audiometer with half octave pitches. For scientific investigation, an instrument with sweep range is best. The half octave audiometer will detect progressive deafness better than one in which the intervals are too long. For clinical purposes, the audiometer with sweep range may be inaccurate in the hands of people not highly trained.

DR. EDMUND P. FOWLER, New York: It is also harder to calibrate.

C. C. BUNCH, Ph.D., St. Louis: I use masking in all my audiometer tests of bone conduction and in those of air conduction when the difference between the two ears is 40 decibels or more. In my own work it is absolutely necessary.

Several types of masking are described in a report published by the Committee on Bone Conduction. I use a buzzer with an intensity of approximately 80 decibels, which can be stepped down. In cases requiring more, I use the Bárány buzzer.

DR. EDMUND P. FOWLER, New York: If the subject gets over 40 or 50 decibels in the air conduction receiver, he gets some hearing of the sound on the opposite side. I use a double stethoscope connected to a pressure tank. If this masks a bone conduction receiver or a fork on the vertex, the ear piece is taken out of the ear to be tested and the bone conduction obtained from the mastoid as usual.

DR. ARTHUR W. PROETZ, St. Louis: Ordinarily I take bone conduction only in the middle range.

C. C. BUNCH, Ph.D., St. Louis: I take it only at 512 and then principally as a check on the Weber and the Rinne test. The devices I have checked were of no value above 1024. Normal persons cannot tell whether they hear by air or bone. At lower tones, tactile sense is confused with hearing. The same observation applies to tuning forks.

DR. EDMUND P. FOWLER, New York: My own bone conduction receiver works satisfactorily up to 4000 double vibrations. Bone conduction is no more mysterious than air conduction.

DR. ARTHUR W. PROETZ, St. Louis: I think the best audiogram is the simple logarithmic curve. One should avoid audiograms with details difficult of reproduction. One should be able to put eight audiograms on a page. I advise leaving off the auxiliary bone conduction lines. They are confusing in print. My own audiogram is legible and simple. I think it best to use the old fork arrangement, "32 - 64 - 128," etc.

C. C. Bunch, Ph.D., St. Louis: There is no agreement as to the value of dropping 64 double vibrations. I prefer to have this value included.

DR. EDMUND P. FOWLER, New York: At 64 double vibrations and lower, the patient does not know whether he is hearing or feeling. If one checks by the loud-

ness balance technic, one finds the threshold so obtained erroneous. The examiner can get all the information he needs, i. e., that there is diminished hearing for low tones, by using 128 or 256 double vibrations.

MR. HOWARD A. CARTER, Chicago: The 64 double vibration tone makes the audiometer more expensive. If the tone is not pure, the patient might hear a higher harmonic.

DR. EDMUND P. FOWLER, New York: One can get a satisfactory examination at 128 to 8000 double vibrations. Patients become tired from a long examination, and this will interfere with the accuracy of the result.

DR. MAX A. GOLDSTEIN, St. Louis: There is such a thing as a conductive defect for the 64 double vibration fork but hearing for 128 double vibrations.

DR. EDMUND P. FOWLER, New York: I have never seen conduction deafness limited to the 64 frequency.

DR. MAX A. GOLDSTEIN, St. Louis: I shall be glad to send you any number of patients who have this type of defect.

C. C. BUNCH, Ph.D., St. Louis: An arbitrary percentage standard for a pure tone has been set up. No one knows just how pure a tone should be for hearing tests. The standard has apparently been satisfactory.

QUESTION: How well standardized are audiometers today? At the Illinois Eye and Ear Infirmary a Maico and a Western Electric do not agree on the threshold tests; the Western Electric does not agree in pitch with the magnesium tuning forks; a Maico and a Sonotone in the same office building vary 15 decibels.

MR. HOWARD A. CARTER, Chicago: If the audiometers were purchased over six months ago they may not agree and should be adjusted.

DR. AUSTIN A. HAYDEN, Chicago: The Maico and the Western Electric are calibrated for a sound-proof room; the Sonotone for a quiet room, allowing about 15 decibels of noise. The starting point is different.

MR. HOWARD A. CARTER, Chicago: The question of pegging the threshold as to energy value should be taken care of at the first possible moment. If there is any question of accuracy, audiometers should be returned to the factory.

DR. EDMUND P. FOWLER, New York: By testing a previously tested person, one can judge of any change. The intensity and purity of tone are not so important.

MR. HOWARD A. CARTER, Chicago: I should say an audiometer should be checked once a year.

DR. FREDERICK T. HILL, Waterville, Maine: Should the audiometer offer high intensities in the upper pitch range?

DR. C. C. BUNCH, Ph.D., St. Louis: Dr. Crowe's report indicates that higher tones are essential for diagnosis. I also refer to Dr. Hill's paper on the use of the monochord. Clinical practice will have to determine how much information will be derived from tests with high tones.

A "sound-proof" room is necessary for detection of slight losses of hearing. It is practical against ordinary city noises.

DR. HORACE NEWHART, Minneapolis: The sound-proof booth reduces extraneous noise about 20 decibels and does very well for clinical use.

DR. EDMUND P. FOWLER, New York: The decibel is a measure in electrical energy and may be considered as the smallest change in intensity perceptible by the ear. It is the standard unit of measure for sound. The energy required at the threshold of normal hearing would be 0 decibels. One decibel would represent the smallest intensity perceptible above that. The average normal threshold was obtained by testing a large number of apparently normal ears.

HARVEY FLETCHER, Ph.D., New York: At about 120 or 130 decibels, sound becomes painful. This represents the upper limit of hearing. At 64 double vibrations there is only half that range, i. e., it takes 40 or 50 decibels more sound measured in physical units to make the ear hear at 64 than it does at 2000.

DR. C. C. BUNCH, Ph.D., St. Louis: Little change for high tones is caused by the opening of the door of a sound-proof room. The low tones have less loss than the middle ones, in which there is 20 to 35 units' loss, especially at 1024. To deduct 15 decibels throughout the range would not take these inequalities into consideration.

DR. GORDON BERRY, Worcester, Mass.: Dr. Bunch uses only 512 double vibrations for testing bone conduction. I use the whole range. It helps in contrasting air and bone conduction and in considering hearing aids.

DR. WERNER MUELLER (by invitation), Boston: The monochord helps one to get a graphic record and has a higher frequency range than forks.

DR. C. STEWART NASH, Rochester, N. Y.: It also has the factor of uniformity.

DR. WERNER MUELLER, Boston: Do audiograms taken by different people on different instruments agree?

DR. C. STEWART NASH, Rochester, N. Y.: No, they represent more of a standard than individual tuning fork tests.

DR. AUSTIN A. HAYDEN, Chicago: The intensity of the tuning fork is not under the control of the operator. The intensity of the audiometer is and can be regulated up or down to determine the threshold.

DR. EDMUND P. FOWLER, New York: By moving the fork quickly closer to the ear, the sound may be made to reappear.

DR. MAX A. GOLDSTEIN, St. Louis: The vibrations from the tuning fork die out as they go along. Unless the fork is electrified, how can definite intensity be maintained?

DR. C. C. BUNCH, PH.D., St. Louis: There has been no standardization of tuning forks. There is an excellent opportunity at this time, provided it is done by a board set up by those with otologic interests.

DR. EDMUND P. FOWLER, New York: Loss at 4096 double vibrations for a piano tuner who must tune by listening to that frequency constitutes a disability. It won't interfere with his hearing of speech.

DR. MAX A. GOLDSTEIN, St. Louis: Among seventy-five individual otolaryngologists in St. Louis, there are eight audiometers. The cost of the instruments is too high.

DR. WALTER P. WHERRY, Omaha: An instrument for documenting hearing must be provided. It costs about $50 to put in a pure 64 cycle; then it is difficult to get a pure 256, and so on. The expected saturation point is 4,000 to 4,500 audiometers. Audiometers formerly cost $600, now $240. With production approaching maximum, the price can be reduced still more provided two standards are maintained: one for research audiometers and one for those used in clinical work. The otologist must support the effort to provide a working usable standard.

From the standpoint of the courts, the average tuning fork test as done by some otologists is a joke.

It costs about $100 to build an audiometer, plus sales and other overhead expense. If manufacturers have other lines, such as hearing aids, the costs can be reduced.

Hearing Aids

DR. ARTHUR W. PROETZ, St. Louis: The worst feature of hearing aids today is bulk.

DR. C. STEWART NASH, Rochester, N. Y.: Manufacturers cannot afford to sell only on the recommendation of otologists. Trying out an instrument in the physician's office is inefficient, and when trying one out on a rental basis the patient gets too many opinions from disgruntled friends. The dealers cannot assume the risk of the second hand problem.

DR. GORDON BERRY, Worcester, Mass.: I agree with Dr. Nash. An instrument should be tried at home, in noisy surroundings and with a single voice at a distance, as in an auditorium.

DR. JOHN H. FOSTER, Houston, Texas: No one except a person with uncomplicated moderate otosclerosis can tell whether a hearing aid will be satisfactory without the trial suggested by Dr. Berry.

DR. GORDON BERRY, Worcester, Mass.: When one can write and have filled a prescription based on the audiogram, the trial may be unnecessary. This is now sometimes successful. The trial and error system requires patience and endurance.

DR. EDMUND P. FOWLER, New York: In obstructive deafness one can prescribe from an audiogram. In nerve deafness it is difficult.

DR. C. STEWART NASH, Rochester, N. Y.: A patient with long bone conduction may hear still better with an air conduction receiver. The fitting of hearing aids is still in the trial and error stage.

C. C. BUNCH, PH.D., St. Louis: The Bureau of Standards could classify instruments in terms of physical characteristics but could not control advertising claims. The Council on Physical Therapy can handle the situation better.

DR. HARRIS P. MOSHER, Boston: What is the cost of servicing?

DR. GORDON BERRY, Worcester, Mass.: The people who manufacture vacuum tube aids service their instruments one year.

QUESTION: What about a salesman referring a customer to an otologist for an audiogram and canal impression, the salesman paying the fee?

DR. C. STEWART NASH, Rochester, N. Y.: I think a patient should have a complete otologic examination, not merely an audiogram, and I should educate the salesman to know the difference. The otologist should, e. g., (1) rule out polyps and acoustic tumors; (2) make an audiometric examination; (3) make a general functional hearing test, plus an examination with tuning forks, and (4) provide honest advice from some one with nothing to sell.

DR. FREDERICK T. HILL, Waterville, Maine: When a physician recommends an instrument and the salesman refers the patient back for examination and the fitting of the ear piece, is it ethical and advisable that the salesman pay the fee?

DR. GORDON BERRY, Worcester, Mass.: If one strives honestly and openly to serve the interests of his patient, he cannot go far wrong. The physician must avoid any tie with the agent which would prejudice his service or advice.

MR. HOWARD A. CARTER, Chicago: If the Council learns of any unethical practices in relation to the sale of any hearing aid, it will be obliged to take the instrument off the accepted list.

DR. HARRIS P. MOSHER, Boston: Why cannot the physician do something for the patient without an extra charge?

DR. GORDON BERRY, Worcester, Mass.: The fitting of the ear piece is an extra service requiring nearly an hour. If the patient cannot afford it, there is no charge.

QUESTION: Is the salesman qualified to select an instrument?

DR. C. STEWART NASH, Rochester, N. Y.: He does it by the trial and error method.

DR. GORDON BERRY, Worcester, Mass.: And if the buyer doesn't like it, his only recourse is to try another.

DR. EDMUND P. FOWLER, New York: When the otologist helps select an instrument, he does not limit himself to one make. The salesman does.

C. C. BUNCH, PH.D., St. Louis: If one knows the amplifying characteristics of the hearing aid and is familiar with the patient's audiogram, one should be able to select a proper aid.

DR. FREDERICK T. HILL, Waterville, Maine: In a case of combined deafness, can you determine objectively whether to use an air or a bone conduction receiver?

DR. EDMUND P. FOWLER, New York: One cannot diagnose combined deafness when bone conduction is normal or prolonged.

DR. KENNETH M. DAY, Pittsburgh: I have mixed deafness—good bone conduction for low tones, not for 1024 to 2048—and hear better by air conduction.

DR. EDMUND P. FOWLER, New York: Patients with a drop in middle and high frequencies usually get along better with an air conduction hearing aid.

DR. GORDON BERRY, Worcester, Mass.: Dr. Day and I speak from personal experience. We have prolonged bone conduction but get better results from air conduction receivers. Music is distorted in the bone conduction receiver.

DR. FREDERICK T. HILL, Waterville, Maine: When should a hearing aid be recommended?

DR. GORDON BERRY, Worcester, Mass.: When the hearing is so low as to handicap the patient. Lip reading is always of service, even to people who hear well. The carbon aids serve better for lesser degrees of impairment, while the vacuum tube aids are better in greater impairment, especially for the higher tones.

DR. HARRIS P. MOSHER, Boston: What is a reasonable price for a hearing aid?

DR. FREDERICK T. HILL, Waterville, Maine: Who can be helped?

C. C. BUNCH, PH.D., St. Louis: The Zenith, a desk instrument with a wall plug, is approved and sells for $29.95. People with loss over 35 decibels and less than subtotal usually get benefit from hearing aids. Marked dips in the hearing not shown by the octave audiometer will prevent good results. Persons with perceptive deafness can also be benefited. There is no evidence that auditory acuity has been improved. Mental capacity may be. There is no nerve regeneration.

DR. FREDERICK T. HILL, Waterville, Maine: Will the use of an aid eventually improve or impair hearing?

DR. EDMUND P. FOWLER, New York: Neither. An intense amplifier used over a long time may be harmful, especially for the hearing of high tones, by reason of the acoustic shock entailed.

DR. GORDON BERRY, Worcester, Mass.: I think there is some danger from thunder clap.

HARVEY FLETCHER, PH.D., New York: I think the danger to a deafened ear is not as great as that to a normal ear. The sound can't go over the capacity limit of the aid, and the ear piece helps to block vibrations from entering directly.

Insufficient evidence has been submitted to substantiate the theory of regeneration of the nerve by external stimulus. The Council would require a group for controls.

DR. GORDON BERRY, Worcester, Mass.: Bone conduction receivers may be used on either side, interchangeably. With an air conduction receiver, the poor ear is used if practicable, to free the better ear for unaided hearing. Sometimes a binaural ear piece is used. A 20 per cent gain is alleged.

DR. EDMUND P. FOWLER, New York: In binaural pure nerve deafness the air conduction receiver is generally more useful on the better ear. If a bone conduction receiver is used, it is best to keep changing sides, to avoid continued pressure on one place.

DR. GORDON BEERY, Worcester, Mass.: A hearing aid should be adjusted so that it is not noisy and confusing.

DR. FREDERICK T. HILL, Waterville, Maine: What aid is small, invisible when stuck in the ear and productive of perfect hearing (referring to some advertising)?

DR. EDMUND P. FOWLER, New York: None.

DR. GORDON BERRY, Worcester, Mass.: A cotton drum soaked in oil to close a perforation helps somewhat.

DR. FREDERICK T. HILL, Waterville, Maine: What type of nerve deafness is aided most by the fistulization operation? How many decibels are gained, and can one get similar improvement from hearing aids?

DR. EDMUND P. FOWLER, New York: The type of deafness, if any, most aided by the fistulization operation is the pure conductive. At present 20 or 30 decibel gains are obtained temporarily. I have no data on cases in which operation has

been done long enough ago to give a percentage on maintained improvement. There must be repeated accurate tests over two or three years. None of the patients who have had the operation and who have been examined by me so far has maintained the improvement. There is no reason why one cannot gain as much, and more, from a hearing aid as from fistulization.

Dr. C. Stewart Nash, Rochester, N. Y.: Eleven hearing aids have been accepted by the Council. I cannot say which is preferable.

Dr. Harris P. Mosher, Boston: For some years I have been asking, "What are the reliable audiometers and the reliable hearing aids?" Today the members of this society have gained something definite. I want to thank the gentlemen who had the courage to express their convictions. Mr. Carter has been a tower of strength.

Book Reviews

The Handbook of Hereditary Diseases, Volume 6. (*a*) Hereditary Deafness and Its Diagnosis. By M. Schwarz, M.D., Professor and Demonstrator in the Ear, Nose and Throat Clinic of the University of Frankfort on the Main. (*b*) Physical Anomalies. By Hellmut Eckhardt, M.D. Pp. 373, with 162 illustrations. Price: paper, 24 reichsmarks; bound, 26 reichsmarks. Leipzig: Georg Thieme, 1940.

This volume is composed of two sections. The first, by Dr. M. Schwarz, deals with hereditary deafness, while the second, by Dr. Hellmut Eckhardt, discusses various anomalies of development of the skeleton and extremities.

In the first half of his monograph, Dr. Schwarz considers such general factors as the embryology and anatomy of the ear. He outlines the physiology of the internal ear, both from the vestibular and the cochlear standpoints. In addition, he describes the various hearing and labyrinthine tests and the relative significance of each.

The second half of the monograph deals exclusively with the problem of deafness, both hereditary and acquired. All current theories on the relative influence of the present known factors are cited and discussed in great detail. A number of cases are reported wherein deafness has been traced back for several generations, and the author is able to show both from his own work and from that of other investigators that deafness as a definite hereditary characteristic can be carried through a number of generations and appear more or less in accordance with a mendelian ratio. He discusses various anomalies in addition to deafness that occur in and about the ears, such as atresia, absence of the auricle or microtia. These too have been traced through a number of generations.

In his monograph, Dr. Eckhardt discusses many anomalies of the fingers, toes, hands, feet and other skeletal structures. He too was able to trace some of these through one or more generations.

This book is of considerable anatomic interest, but its value to the practical otologist is questionable. However, as a reference book it is extremely useful, since it has a condensed summary of all the present day theories concerning hereditary deafness.

A Manual of Otology, Rhinology and Laryngology. By Howard Charles Ballenger, M.D., Assistant Professor of Otolaryngology, Northwestern University School of Medicine, Chicago. Pp. 302, with 90 engravings and 4 color plates. Price, $3.75. Philadelphia: Lea & Febiger, 1940.

The author offers this manual as a small, concise textbook on diseases of the nose, throat and ear, emphasizing anatomy, causes, symptoms and diagnosis but including only the usually accepted general and local treatments.

He has compressed into 302 pages considerable material, utilizing the format, print, sketches, diagrams, etc., of his larger work, the seventh edition of "Diseases of the Nose, Throat and Ear" by Drs. W. L. Ballenger and H. C. Ballenger.

This manual is excellent and should be of greatest usefulness to the undergraduate student, who will profit much by frequent reference to it. It probably will not find many readers among specialists or graduate students in otorhinolaryngology, for whom the larger textbooks, of which there are several high grade standard ones are the more suitable.

It is not amiss to stress once more the teaching value of this manual as well as the excellence of the entire format, for the author has well succeeded in his purpose.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Cleveland. Time: June 2-6, 1941.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank Brawley, 30 N. Michigan Ave., Chicago.
President-Elect: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 19-24, 1941.

American Broncho-Esophagological Association

President: Dr. Gabriel Tucker, 250 S. 18th St., Philadelphia.
Secretary: Dr. Paul Holinger, 1150 N. State St., Chicago.
Place: Cleveland. Time: June 3, 1941.

American Laryngological Association

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Place: Atlantic City, N. J. Time: May 28-30, 1941.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Ambassador Hotel, Los Angeles. Time: June 16-18, 1941.

SECTIONS:

Eastern.—Chairman: Dr. N. S. Weinberger, Robert Packer Hosp., Sayre, Pa.

Southern.—Chairman: Dr. William G. Kennon, Doctors Bldg., Nashville, Tenn.

Middle.—Chairman: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago.

Western.—Chairman: Dr. Robert C. Martin, 384 Post St., San Francisco.

American Otological Society

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. Isidore Friesner, 36 E. 73d St., New York.
Place: Atlantic City, N. J. Time: May 26-27, 1941.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.